

# Nutritional Strategy for Adolescents Undergoing Bariatric Surgery: Report of a Working Group of the Nutrition Committee of NASPGHAN/NACHRI

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## ABSTRACT

Surgical options for the treatment of adolescent obesity have been gaining popularity. Adolescent patients present a particular challenge to clinicians, secondary to age-related issues, revolving around both mental and physical growth. These age-related issues require a unique approach to nutritional intervention for adolescents undergoing bariatric surgery as opposed to standardized approaches for adults. Despite the increasing numbers of adolescents undergoing obesity surgery, evidence-based nutritional guidelines have yet to be published. The goal of this document is to provide the clinician with recommendations on how to assess, educate, nourish, and monitor the adolescent who has undergone obesity surgery. A multidisciplinary panel composed of 3 pediatric gastroenterologists, 1 psychologist, and 3 registered dietitians from the Nutrition Committee for the North American Society of Pediatric Gastroenterology, Hepatology, and Nutrition and National Association of Children's Hospitals and Related Institutions, with experience in nutrition and adolescent weight loss surgery, reviewed the medical literature for evidence-based practice for nutritional strategies for patients undergoing bariatric surgery. In addition to this group, an adolescent medicine physician was consulted for matters related to reproductive health. The present article presents a consensus of recommendations based on a review of the literature. In areas for which there was a lack of evidence to support the recommendations, best-practice guidelines were used. The present article provides the clinician with an overview of the nutritional concerns for adolescent patients undergoing obesity surgery. These guidelines address the preoperative educational pathway, the post-operative diet progression, recognition of disordered eating, guidelines for female reproductive issues, and assistance for the adolescent in a school/college environment.

**Key Words:** adolescent obesity, bariatric surgery, nutrition education, nutritional assessment, nutritional deficiency, nutritional guidelines, nutritional needs

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Obesity seen during adolescence is a multifactorial process with several contributing factors, including metabolic dysregulation, genes, environmental/socioeconomic stressors, and energy imbalance (1). Patients with obesity carry an increased morbidity and are at risk for obstructive sleep apnea, metabolic syndrome, orthopedic issues, hypertension, type 2 diabetes, and nonalcoholic steatohepatitis (1,2). These comorbid conditions affect an adolescent's quality of life and daily functioning (3). The obesity rates of children ages 2 to 19 are now 17.6%, with obesity being classified as a body mass index (BMI) above the 95th percentile (4). Obese adolescents have a 50% to 77% risk of becoming obese adults with an increase to approximately 80% given 1 obese parent (1,5). In view of this, surgical treatments for pediatric obesity have increased in popularity.

Although many weight management centers perform weight loss surgery (WLS) for the treatment of adolescent obesity, no standard for nutritional management exists for these patients. The present article describes recommendations from a multidisciplinary panel with a background in adolescent nutrition and WLS. The North American Society of Pediatric Gastroenterology, Hepatology, and Nutrition and National Association of Children's Hospitals and Related Institutions (NASPGHAN-NACHRI) Bariatric Nutrition Subcommittee included 3 pediatric gastroenterologists, 3 registered dietitians (RDs), and 1 psychologist with expertise in bariatric nutrition. The content of the article was initially outlined by the panel. Articles written in English were identified using PubMed and MEDLINE searches. No exclusions were made based on publication date. All of the human studies related to bariatric surgery and nutrition were reviewed. Additional articles identified by subcommittee members were also reviewed. A total of 417 articles related to bariatric surgery and nutrition were reviewed including case series and reports. Using the best available evidence from the literature, content was then evaluated and discussed. The subcommittee conferenced on 8 separate occasions; it based its recommendations on its study of the literature review combined with expert opinion and evidence available in the adult literature when pediatric evidence was insufficient. The present article is intended to be a guide for the clinicians involved in the nutritional care of obese adolescents undergoing bariatric surgery.

Nutritional recommendations for the treatment of the adolescent undergoing WLS can be classified into 4 areas: nutritional assessment, education, nutritional needs, and monitoring of nutritional status. The role of the RD is considered an integral component of a multidisciplinary WLS team. It is recommended that an RD with a certificate in pediatric weight management and/or experience in WLS clinically evaluate, educate, and monitor the nutritional status of the adolescent undergoing WLS.

## PREOPERATIVE NUTRITIONAL ASSESSMENT

Before any WLS procedure, all adolescents should undergo an appropriate nutritional evaluation, including selective micronutrient measurements to identify nutritional and educational needs. Nutrition and meal planning guidance should be provided to the patient and family before WLS and during the postoperative hospital course and reinforced during future outpatient visits. In comparison with purely restrictive procedures, more extensive preoperative nutritional evaluations are required for malabsorptive procedures. A clinical session should be arranged with an RD who is a member of the WLS team. Before surgery some candidates may already have micronutrient deficits due to nutrient-poor food choices, lack of dairy, and low fruit and vegetable and/or whole grain intake. Listed in Table 1 are the recommended laboratory parameters to be considered as part of the nutritional evaluation. If preexisting nutritional deficiencies are identified in the initial assessment, then a dietary intervention to correct these deficiencies should be advised to the adolescent. For all adolescents, a preoperative multivitamin regimen should be put into place because of the high prevalence of micronutrient deficiencies in morbidly obese patients and to correct possible preexisting micronutrient deficiencies.

A food frequency/eating behavior questionnaire completed by the adolescent may provide additional nutritional information. A healthy balanced diet consisting of adequate protein, fruits, vegetables, and whole grains is advised during the preoperative period to assist in weight stability or weight loss. In addition, behavioral strategies for portion control are encouraged such as eating from small plates and using small utensils. Calorie needs based on height, ideal body weight, and age should be determined. Estimated energy, protein, and fluid intake as well as meal frequency patterns should be reviewed. Assessment of mindful eating habits may be of value in examining the underlying understanding, attitudes, and circumstances that lead to healthy eating patterns necessary for successful food choices.

A variety of factors influence food accessibility and food choices that ultimately influence outcome after WLS. Review of the family, family support, and home environment should be considered to be part of the initial assessment. The “family” for the adolescent requires definition because this may not be at the permanent address, but rather focus on the locations where significant time is spent routinely and where food/meals are available for consumption. All caregivers within the “family” network should be invited to attend any nutrition education sessions and participate in development of a sound home environment. The family should provide an environment that is nutritionally supportive of weight loss. This is defined as limited energy-dense and nutrient-poor

foods replaced by nutrient-dense foods. In the family in which there are members who are of varying body sizes (ie, healthy weight parent[s] or sibling[s]), the family should be encouraged to provide healthy foods for the entire family. In addition, there is evidence that supports limited eating out and family meals in the adolescent’s household to support weight management (6,7). Because of the greater independence of youths during the adolescent period, education regarding change in health behaviors should be directed to the adolescent. Consideration also needs to be given to striving to make change within the entire family because the parents or caregivers are often the individuals making the food purchasing decisions and supporting the adolescent undergoing bariatric surgery. The nutrition interview should include questions to ascertain the level of familial support. If families are having difficulty in providing a supportive environment for the adolescent, then a psychology consult should be considered. Family culture affects food preferences and availability. Culture may also change the interaction between the family and the traditional foods that are in the household. It is important to integrate dietary recommendations within a culturally sensitive framework because this may improve adherence (8–10).

It is important to note differing parenting styles in working with families of adolescents undergoing bariatric surgery. Type of parenting style may affect adolescent functioning and likely plays a role in how families approach nutritional counseling around bariatric surgery. Baumrind (11) developed 4 models of parenting: authoritarian, authoritative, permissive, and disengaged. Authoritarian style is demonstrated via strict rule setting and abidance. These adolescents may be best motivated by a rule-setting approach. The authoritative style leads to parents being warm and involved, yet consistent and firm in limit setting. Permissive parents are those that are accepting and impose few rules and restrictions, so these adolescents may have difficulty around rule setting and may test the guidelines established by the clinician. Disengaged parents often present as disinterested and may not provide the support to help the adolescent with adherence. For those adolescents, reliance on the family to make change may be difficult; change may need to lie with the adolescent. Each one of these parenting styles may result in a specific set of behaviors demonstrated by the adolescents. Acknowledgment of these parenting styles may assist in optimizing educational outcomes.

## NUTRITION EDUCATION

The preoperative pathway should be standardized to ensure that all adolescents receive the same information. Because of differing learning styles, information may need to be presented in a variety of different teaching methods. The initial visit with the

TABLE 1. Considerations for nutritional screening

Nutrient deficiency	Prevalence in adults	Screening laboratories
Thiamin (B <sub>1</sub> )	15%–29% (more often in African Americans/Hispanics)	Blood thiamin level, erythrocyte thiamin transketolase, or transketolase urinary thiamin excretion
Cobalamin (B <sub>12</sub> )	10%–13%	Serum homocysteine level, urine/serum methylmalonic acid level, and/or serum vitamin B <sub>12</sub> level
Iron	9%–16% of women (risk factors girl, anemic/heavy menses, low dietary intake)	Serum iron, serum ferritin, total iron-binding capacity
Vitamin D	60%–70% (risk factors sedentary, indoors, darker skin)	25-Hydroxy vitamin D level
Folate	20% (risk factors high-fat diets)	Serum homocysteine level and/or serum folate or red blood cell folate level

RD should include the previously mentioned assessment of nutritional status and current intake. Education regarding basics of WLS postoperative nutrition principles should be included in this initial visit as well. It is recommended that the clinician establish nutritional goals together with the adolescent. These goals should be shaped around the principles that are similar to the postsurgical nutrition plan, such as elimination of sugared beverages, consuming 3 or 6 meals per day (depending on the procedure), and taking vitamins as directed.

Adolescents may present varied exposure to “dieting.” It should not be assumed that they are familiar with or knowledgeable about what constitutes a healthy lifestyle. The RD should consider standardized preoperative teaching regarding healthy eating to include appropriate serving sizes and food quality according to the dietary guidelines. During the preoperative phase, it is suggested that the adolescents be encouraged to maintain some method of self-monitoring such as food records, recording healthy foods that they are eating because self-monitoring has been documented to assist in weight maintenance (12). These food records can also be used as a way to educate the adolescent on how to quantify the protein in their current diet to provide familiarity with this concept in preparation of the postoperative meal planning process. A minimum of 6 preoperative nutrition visits are recommended to complete the nutritional assessment and educational components in preparation for WLS (Table 2).

It is recommended that educational information be provided in verbal and written fashion. The RD may want to consider taste testing or exposure to protein supplements to assist the adolescent in determining acceptability before WLS. Education regarding the soft and regular meal plans may be completed at the postoperative visits as completing this education preoperatively can be difficult for the adolescent to retain. Completion of the liquid and smooth food meal plans preoperatively assists the adolescent in becoming familiar with meal planning.

Because of differing learning styles and family support in the adolescent population, the designated visit number may require some flexibility. The clinician may need to adapt the teaching style to the adolescent before or during the nutrition education process. A consistent framework for education as well as small, agreed-upon goals may assist in the increase of retained information in these adolescents.

### DIET PROGRESSION

The diet advancement protocols for Roux-en-Y gastric bypass (RYGB), vertical sleeve gastrectomy (VG), and laparoscopic adjustable gastric banding (LAGB) will vary by the number of meals per day, the consistency, texture, and progression of the amount of food consumed at a meal (13) (Tables 3–5).

TABLE 2. Recommended nutritional assessment and educational components in preparation for WLS

Initial nutrition assessment
Nutrition educational session regarding food quality choices (possibly in group session)
Nutrition education regarding food quantity choices for preoperative energy needs (possibly in group session)
Nutrition education regarding liquid meal plans
Review of accuracy of liquid meal plans and, if adequate, then nutrition education regarding smooth food meal plans
Review of smooth food meal plans and, if adequate, review of questions/concerns regarding post op meal plans

WLS = weight loss surgery.

The postoperative diet focuses on weight loss with preservation of lean body mass by recommending a diet high in protein (1.0–1.5 g protein/kg ideal body weight), low in simple carbohydrates, free of added simple sugars, and with modest fat intake.

### MACRONUTRIENT NEEDS

#### Hydration

Daily recommended intake (DRI) for girls ages 14 to 18 years is 2.3 L of water per day, increasing to 2.7 L/day when older than 18 years. Boys have a higher fluid requirement with 14- to 18-year-old boys requiring 3.3 L/day; boys older than 18 years have a DRI of 3.7 L/day (14). After bariatric surgery, patients are at risk for dehydration usually because of inadequate intake, vomiting, or diarrhea. Given the nature of restrictive forms of WLS, many patients are not able to take in large volumes of water at 1 point in time and must consume their water in small volumes frequently throughout the day. Fluid requirements can vary for each patient and should be guided by thirst; however, a minimum of 48 to 64 oz of total fluids per day has been recommended in adult bariatric surgery patients (15,16). These liquids may equal 24 to 32 oz of clear liquids plus 24 to 32 ounces of full liquids such as nonfat milk, Lactaid or soymilk, or light yogurt (15). All of the liquids should be decaffeinated and sugar-free with low sorbitol content.

To maintain hydration, strategies for addressing vomiting in a patient postsurgery include adherence to serving size guidelines and chewing food extremely carefully before swallowing; ensuring that adolescents avoid liquid intake 30 minutes before and after meals may also aid in reducing vomiting. Diarrhea associated with dumping syndrome may contribute to dehydration in patients

TABLE 3. Diet advancement protocols for Roux-en-Y gastric bypass, vertical sleeve gastrectomy, and laparoscopic adjustable gastric banding

	Stage 1: ice chips, water, sugar-free clear liquids	Stage 2: full liquids, high protein	Stage 3: smooth consistency foods and liquids	Stage 4: soft foods	Stage 5: all textures—healthy foods
Roux-en Y	1 oz/h for the first 24–48 h, then for 3–7 days ad lib	Wk 2–4	Wk 4–6	Wk 7–9	Begins at wk 9 and for life
Gastric band	1 oz/h for the first 24–48 h	First 2 wk after surgery	Wk 3–4	Wk 5–6	Wk 7 and for life
Sleeve gastrectomy	1 oz/h for the first 24–48 h, then for 3–7 days ad lib	Wk 1–5	Wk 5–8	9–12 wk postop	At wk 13 and for life

TABLE 4. Postoperative diet by stages

	Goals per day
Stage 1: sugar-free clear liquids	4–6 oz of water or sugar-free clear liquids per hour (48–64 oz/day) Acceptable sugar-free clear liquids include water, clear broth or bouillon, sugar-free gelatin, sugar-free fruit-flavored drinks, fruit ice made with sugar-free fruit-flavored drink, sugar-free popsicles Sugar-free clear restrictions: no carbonated beverages, no caffeine, no red dye
Stage 2: high-protein full liquids	Calories: 500–600 kcal Protein: 50–60 g Fluid: 80–90 oz or based on estimated requirements New foods introduced: skim or 1% milk, low-fat soy or Lactaid milk, high-protein drinks, light yogurt (plain or vanilla) thinned out with milk Meal pattern: 3–6 meals per day Volume: 1/2 cup per meal for solid foods
Stage 3: smooth consistency high-protein foods	Calories: 500–700 kcal Protein: 60 g Fluid: 80–90 oz (or based on estimated requirements) New foods introduced: scrambled eggs, blenderized/minced turkey, chicken, flaked fish or mashed tofu, tuna, melted low-fat cheese low-fat cottage cheese (small curd only), low-fat ricotta cheese Consistency of food: smooth Try new foods 1 at a time (1/4 cup) every 2–3 d Meal pattern: 3–4 meals per day Volume: 1/2 cup per meal for solid foods or 5–6 oz per meal of protein drink
Stage 4: soft foods—other protein foods, fruit, vegetables, and grains	Calories: 700–800 kcal Protein: 60 Fluid: 80–90 oz (or based on estimated requirements) New foods introduced: protein foods: shaved delicatessen meats, low-fat cheese, lean pork, cooked beans Fruit: soft or canned in own juice, no skin Vegetables: soft cooked or canned Grains: toast, low-sugar cereal, crackers, oat meal, rice, pasta, mashed potatoes (choose mainly whole-grain products, foods) Meal pattern: 3–6 meals per day Volume: 1/2–1 cup per meal solid foods or 1 cup (8 oz) protein drink
Stage 5: healthy foods for life	Calories 800–900 kcal Protein: 60 g Fluids: 80–90 oz (or based on estimated requirements) New foods introduced: all healthy food choices Meal pattern: 3–6 meals per day Volume: Up to 3/4 to 1 1/2 cup per meal

After filling/adjustments for laparoscopic adjustable gastric banding, usually done 6 weeks after surgery and then every 6 weeks until satiety is reached, a full liquid diet for 1 to 2 days is recommended after fill with advancement to soft solids for 4 to 5 days thereafter as tolerated (1).

undergoing gastric bypass. These adolescents should be instructed to avoid table sugar, candy, honey, jelly, and other concentrated forms of sugar, which may increase osmolarity of contents entering the small intestine with subsequent diarrhea.

## Protein

Although true protein requirements postbariatric surgery are unclear, DRI for the public state that girls older than 13 years have a DRI of 46 g of protein per day. Boys ages 14 to 18 years have a DRI of 52 g/day, increasing to 56 g/day for boys older than 18 years (14). Given the nature of WLS, which aims to restrict exogenous energy

intake, it is not surprising that protein deficiency is seen in as much as 13% of patients after RYGB but is less likely in patients with a Roux limb <150 cm in length (17,18). Protein malnutrition is the most severe macronutrient deficiency seen after pancreaticobiliary diversion (15).

Present studies indicate that patients lose fat mass in relation to fat-free mass at a ratio of 4:1 with restrictive surgeries such as banding or RYGB (19,20). Because the goal of bariatric surgery is to preferentially decrease fat mass, adequate protein intake is imperative to prevent the loss of lean body mass. In adults undergoing bariatric surgery, the recommendation is an intake of at least 60 to 90 g of protein per day for RYGB and 80 to 120 g/day for patients with a biliopancreatic diversion (15,21,22). Because many

**TABLE 5.** Dietary tips for adolescents undergoing weight loss surgery

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Avoid high-sugar foods and/or fat content to prevent “dumping syndrome.” Symptoms may include cramping, clammy feeling, sweating, heart racing, vomiting, and/or diarrhea. Late dumping may occur up to 2 h after eating foods high in sugar or fat.

Meals should be eaten in approximately 20 min.

Adolescents should be encouraged to stop drinking 30 min before a meal and not to drink until 30 min after a meal. Eating and drinking at the same time may decrease satiety and/or increase incidence of vomiting.

Encourage the patient to eat slowly, taking small bites. Make sure food is cut into small pieces. Chew food well until it has a pureed consistency.

Do not eat or drink past the first feeling of fullness. When feeling full, stop eating and put food away or discard what is left. To prevent dehydration, it is important to encourage the patient to continually sip fluids throughout the day, preferably water. Consider limiting caffeinated products.

Do not lie down within 1 h after meals.

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patients have a difficult time maintaining adequate protein intake, they use protein supplements that are high in protein and low in calories and sugar to reach protein intake guidelines. Protein intake should be quantified periodically by the medical team to ensure adequate education and implementation of the prescribed diet. Parenteral supplementation should be considered in patients with severe protein malnutrition that is not responsive to oral protein supplements (15). Given the current practices in the pediatric population, 60 to 90 g of protein per day is recommended in adolescents undergoing RYGB, lap band, and gastric sleeve procedures.

## Energy Needs

Total energy expenditure should be calculated based on the clinical center’s preestablished route of ascertaining energy expenditure to elicit weight loss. This value should be reassessed with significant changes in weight or when expected weight loss is not evident. Recent use of Bod Pod, an air displacement plethysmograph, that uses whole-body densitometry to determine body composition (fat and fat-free mass), may be a useful tool in both adults and children in determining total energy expenditure.

## MICRONUTRIENT NEEDS

Vitamin and mineral deficiencies are not uncommon in Westernized diets. The 2 most common deficiencies noted are iron and vitamin D. Because obesity itself is a malnourished state, there is increased risk of these as well as other vitamin and mineral deficiencies. This risk of micronutrient deficiency can be compounded by bariatric surgery. It is important to address these potential deficiencies before any bariatric procedure. Minimal preoperative laboratory studies should include evaluation of the more common deficiencies found including vitamin D and calcium; complete blood cell count with iron, transferrin, and ferritin, red blood cell folate (homocysteine level recommended in some centers for folate monitoring), vitamins B<sub>1</sub>, B<sub>6</sub>, B<sub>12</sub>, and 25-hydroxy vitamin D level should be obtained. Supplementation with a

complete multivitamin should occur in all candidates undergoing bariatric procedure. For those adolescents noted to have a specific micronutrient deficiency, correction of the deficient state should occur before the bariatric procedure.

Vitamin and mineral supplementation is important after bariatric surgery to avoid sequelae from nutrient deficiencies. Attention to the type of supplementation is important as studies report differences in vitamin and mineral content. In the case of iron, over-the-counter supplements can range from 0% to 230% of the labeled iron content; generic brands (36%) had less than commercially labeled amounts (81%). Specially formulated vitamin supplements contained 183% and 230% of reported iron amount (23).

## Vitamin B<sub>1</sub> (Thiamin)

Thiamin is a coenzyme in the oxidation of  $\alpha$ -keto acids and 2-keto sugars, which function in pyruvate metabolism and synthesis of acetylcholine. Thiamin deficiency leads to accumulation of lactate and alanine, and reduced high-energy phosphate synthesis (24). There are minimal stores present in the body; total body thiamin deficiency can occur within 18 days following dietary restriction (25). Thiamin is actively absorbed in the jejunum and ileum but can be taken up via passive diffusion at higher concentrations (26).

Moderate-to-severe thiamin deficiency has been previously found in 10% to 20% of obese women (27). High-carbohydrate diets have been associated with thiamin deficiency (28). After WLS, thiamin deficiency appears most likely to occur in patients who experience persistent vomiting and/or excessive weight loss after surgery (29). Among adults, after gastroplasty and gastric bypass, thiamin levels are below normal in 50% of patients, often accompanied by symptoms of thiamin deficiency (30). The incidence of acute postgastric reduction surgery neuropathy has been estimated at 5.9/10,000 adult WLS cases; common symptoms include weakness, hyporeflexia, and vomiting (31). In adolescents, thiamin deficiency has been reported in girls after gastric bypass surgery presenting 4 to 6 months after bypass with increasing lower extremity weakness, pain in the lower extremities in stocking distribution, nystagmus, and hearing loss (32).

Current published DRI for thiamin is 1.2 mg/day for boys ages 14 to 18 years and 1.0 mg/day for girls ages 14 to 18 years (33,34). Adolescents who have undergone bariatric surgery should be receiving a minimum of 50 mg of thiamin per day, which can be supplied by including an additional B complex in the vitamin regimen. For patients with significant protracted emesis, addition of thiamin to parenteral nutrition during the postoperative period is recommended (29).

## Vitamin B<sub>6</sub> (Pyridoxine)

The DRI for vitamin B<sub>6</sub> is 1.2 mg/day for girls ages 14 to 18 years and 1.3 mg/day for boys ages 14 to 18 years (33,34). In adults after gastroplasty, intake of vitamin B<sub>6</sub> at current adult girl DRI (1.6 mg) is inadequate to maintain coenzyme saturation of erythrocyte aminotransferases (35). It is recommended that individuals undergoing WLS take a multivitamin that includes vitamin B<sub>6</sub>.

## Vitamin B<sub>9</sub> (Folic Acid)

Folic acid is found in fortified breakfast cereals, leafy green vegetables, and liver and kidney. It requires hydrolysis to allow jejunal absorption and serves as a cofactor in the single carbon atom transfer needed for amino acid metabolism, purine/pyrimidine

syntheses, and generation of S-adenosylmethionine. Deficiency in folic acid consequently results in elevation of serum homocysteine levels. Low levels of folic acid found in 7% of obese women have been attributed to the consumption of a high-carbohydrate diet (36). One year after WLS in adults, increased homocysteine levels were found in 32% to 66% of patients attributed to decreased folic acid (37).

Among teens, the DRI for folic acid for both boys and girls is 400  $\mu\text{g}$  daily (33,34). Recommended supplementation after bariatric gastric bypass or biliopancreatic diversion based on adult experience is between 400  $\mu\text{g}$  and 1 mg daily (38). Because folic acid is absorbed throughout the small intestine, a multivitamin containing folic acid should suffice for restrictive procedures such as LAGB.

### Vitamin B<sub>12</sub> (Cobalamin)

Cobalamin found in fish, milk, eggs, meat, and poultry serves as a substrate for cofactors of enzymes including methionine synthase, glutamate mutase, dioldehydratase, and methyl malonyl coenzyme A mutase. It is cleaved by gastric acid and released from salivary R-protein by proteases. It then binds to intrinsic factor and goes to the ileum where it is released, and migrates from the brush border to transcobalamin proteins.

Vitamin B<sub>12</sub> deficiency develops in restrictive and malabsorptive operations but rarely arises after gastroplasty or banding unless there is decreased intake. Two-thirds of gastric bypass patients in 1 series had low vitamin B<sub>12</sub> levels (39) attributed to the lack of acid production in the pouch (40).

The DRI for cobalamin is 2.4  $\mu\text{g}$ /day for boys and girls ages 14 to 18 years (33,34). For individuals undergoing malabsorptive/restrictive procedures, supplementations using the 500- $\mu\text{g}$  sublingual form or monthly injections are recommended. Recommended treatment for vitamin B<sub>12</sub> deficiency in adult bariatric patients is recommended as oral (350  $\mu\text{g}$  daily), 1000  $\mu\text{g}$  im q-2 to q-3 months or 500  $\mu\text{g}$ /week nasal spray.

### Vitamin A

Fat-soluble vitamin deficiencies have been reported after bariatric surgery, more commonly after biliopancreatic diversion, which alters fat mixing with pancreatic enzymes and bile salts more than with gastric bypass. In an adult series, vitamin A deficiency has been reported in 10% of patients after bypass surgery manifested as night blindness (39). The DRI for vitamin A is 900  $\mu\text{g}$ /day for boys ages 14 to 18 years and 700  $\mu\text{g}$ /day for girls ages 14 to 18 years. Supplementation with vitamin A at the current DRI is recommended for adolescents postbariatric surgery. This amount is usually found in a daily multivitamin. For individuals undergoing more extensive malabsorptive procedures such as duodenal switch or long-limb gastric bypass, additional supplementation may be required.

### Vitamin D/Calcium

Vitamin D is a prohormone that is essential for the normal absorption of calcium in the gastrointestinal tract. Deficiency in vitamin D leads to hypocalcemia, hypophosphatemia with resultant nutritional rickets in children, and osteomalacia in adults. In adults, vitamin D deficiency has also been linked to cardiovascular disease, insulin resistance, and hypertension. In addition to a number of large case studies, the National Health and Nutrition Examination Survey (NHANES III) has emphasized the high prevalence of vitamin D deficiency in industrialized nations with up to 14% in

the United States (41,42). The resurgence of vitamin D deficiency is likely caused by a number of dietary and environmental factors, including BMI, milk ingestion, and sun exposure (43).

Calcium can be absorbed by all segments of the small intestines, although the duodenum and jejunum are most active. Vitamin D is absorbed principally in the jejunum. Data on vitamin D status after bariatric surgery have shown mixed results (29). It does appear that vitamin D deficiency is more common in malabsorptive/restrictive procedures than in restrictive procedures alone. Given the predisposing factor of obesity in developing vitamin D deficiency, it is believed by most adult bariatric centers that patients undergoing bariatric surgery are at risk for developing bone mineral density and metabolism issues after procedure (29,44). Although there are no current national guidelines for adults undergoing bariatric surgery in regard to calcium and vitamin D supplementation, many bariatric centers provide supplements (38).

The DRI for vitamin D and calcium is 400 IU/day and 1300 mg/day, respectively, for boys and girls ages 14 to 18 years (33,34). Bariatric programs that performed bariatric surgery on adolescent patients were surveyed; most prescribed calcium with vitamin D in the form of calcium citrate or calcium carbonate with vitamin D. The minimum recommended dose of calcium is 1300 mg/day; the minimum vitamin D intake should be 600 IU (45). Some pediatric programs prescribe up to 2000 IU/day. If vitamin D deficiency is detected, supplementation of 1000 to 5000 IU may want to be considered for correction of deficiency (46).

### Iron

Although the prevalence of iron deficiency among 1-year-old infants in the United States has declined because of improved iron supplementation during the first year of life (47,48), the rate of iron deficiency in older children and adolescents has remained relatively unchanged during the last 4 years (49). Adolescent girls who are obese have a higher risk of developing iron deficiency than their nonobese counterparts (50). The DRI for iron in boys 14 to 18 years is 11 mg daily and 15 mg daily for girls 14 to 18 years (33,34).

Iron deficiency has been reported in 15% to 50% of adults after gastric bypass, with similar numbers with biliopancreatic diversion secondary to the decreased absorption from achlorhydria or because of the use of acid suppression therapy coupled with decreased intake/tolerance of iron-rich food (51,52). Iron deficiency rarely occurs after gastroplasty or gastric banding, but mild iron deficiency has been reported in long-term series secondary to meat intolerance (53). To prevent iron deficiency in gastric bypass or biliopancreatic diversion, treatment with ferrous sulfate 300 mg daily with vitamin C has been advocated by (51). A multivitamin containing iron should suffice for adolescents undergoing restrictive bariatric procedures if they develop mild meat intolerance. With individuals undergoing restrictive procedures who are ingesting a diet including a good source of iron, which include liver, beef, whole-grain breads, cereals, eggs, and dried fruit, supplementation may not be required. It is important to remember that vitamin C can increase iron absorption by 50% (54).

### Zinc and Magnesium

The DRI for zinc is 11 mg daily for boys and 9 mg daily for girls ages 14 to 18 years (33,34). There are minimal reports of zinc deficiency after bariatric surgery aside from alopecia noted after gastric bypass surgery among adults that reversed after zinc supplementation and zinc deficiency noted among bypass patients with severe protein energy malnutrition. Because zinc absorption is

dependent upon fat absorption, patients undergoing biliopancreatic diversion or duodenal switch are at risk for zinc deficiency (55). Zinc deficiency is unlikely to occur in restrictive procedures alone.

Low levels of magnesium in 5% of patients have been documented after BPD, but no significant clinical complications are reported in the literature (56). Although it should be noted that hypomagnesemia can be a cause of recalcitrant hypocalcemia. Recently, the Food and Drug Administration announced a drug safety communication regarding the long-term use of proton pump inhibitors and the development of hypomagnesemia (57). This should be taken into consideration when evaluating bariatric patients, given the high use of acid-blocking medication in this patient population. Zinc and magnesium supplementation are recommended as needed based on clinical suspicion and serum levels.

### Copper

The DRI for copper is 890 µg daily for boys and girls between 14 and 18 years. Copper deficiency has been reported after gastric bypass (58). Among a recent series of patients with development of posterior myelopathy after gastric bypass, 5 of 8 patients were copper deficient (59). In 1 case series it was noted that 25% of subjects with copper deficiency presenting clinically as anemia and neutropenia had undergone bariatric surgery (60). The clinician may want to consider a review of the multivitamin preparation that the adolescent is taking to ensure that it does contain copper, especially if iron deficiency is not easily corrected as this may be caused by copper deficiency (61).

### Omega-3 Fatty Acids

There is evidence to support the role of ensuring adequacy of omega-3 fatty acid intake in adults with cardiovascular disease in particular. The current recommendation of the American Heart Association is a minimum of 8 oz of fatty fish per week for adults. Of note are concerns regarding mercury contamination of fish and/or supplements, but it is believed that the benefits outweigh the potential risks (62). Because of the restrictive nature of most obesity surgery procedures, the quantity of consumption of fatty fish may already be limited in the adolescent undergoing WLS. The clinician may want to consider a careful analysis of the intake of the adolescent to determine adequacy of omega-3 in the diet and educate regarding high omega-3 food choices or supplement to promote nutritional adequacy.

### Other Micronutrients

Although other micronutrient deficiencies can occur especially in restrictive/malabsorptive procedures, they are not as well defined as the aforementioned nutrient deficiencies. Supplementation

with a complete multivitamin daily should suffice for these other micronutrients.

## NUTRITIONAL MONITORING

This risk of developing micronutrient deficiency increases with bariatric surgery. It is important to address these nutritional deficiencies in a timely manner. Additionally, clinicians should consider the long-term nutritional monitoring needs and therefore develop a transitional plan to an adult bariatric care center to provide for long-term monitoring for nutritional deficiencies. Minimal postoperative laboratory studies should include a core set of nutritional laboratories: a complete blood cell count, iron, ferritin, thiamin, vitamin B<sub>12</sub>, methylmalonic acid, 25-hydroxy vitamin D, albumin, calcium, magnesium, phosphorus, red blood cell, and/or serum folate levels semiannually and annually. For those individuals with extensive weight loss nutritional laboratories should be obtained every 3 months. If concerns for osteomalacia exist, consideration of pre- and postprocedure dual-energy x-ray absorptiometry scan may be beneficial. It is important to ensure that a patient's weight does not exceed the machine's capacity because many dual-energy x-ray absorptiometry scans can only accommodate up to 300 lb.

When monitoring micronutrients, it is imperative to use age-appropriate cutoffs to appropriately diagnose and treat nutritional deficiencies. Table 6 addresses age-appropriate laboratory parameters to diagnose iron deficiency anemia. Table 7 addresses additional nutritional parameters to monitor.

Ferritin concentrations are often first decreased when iron intake is inadequate; however, ferritin is not recommended as a screening tool because it can be affected by other issues (eg, inflammatory process).

## OTHER CONSIDERATIONS

### Hunger Assessment

Volume of food consumed is decreased after WLS (65). It is unclear by which mechanism, hormonal or mechanical, food intake is limited. Despite which mechanism may be responsible, it makes sense that recognition regarding hunger/satiety cues would aid in improved outcomes. Teaching hunger satiety cues before and after surgery may assist in recognition of the adolescent for the need for less volume of food after obesity surgery. One method of teaching hunger/satiety is the use of a hunger scale. This may be accomplished by asking the adolescent to record hunger/satiety on a scale of 1 to 10 before and in the middle of a meal (with the number 1 being the number for extreme hunger and 10 being the number for extreme fullness). If they record a number >7 at the middle of the meal, then they should be asked to consider ending the meal. Teaching and encouraging intuitive eating before and after WLS may assist in growing awareness of hunger and satiety and therefore, may limit the volume of food consumed, which may affect overall postsurgical weight loss.

TABLE 6. Fifth percentile cutoffs for measure of iron deficiency in childhood

Population	Hgb, g/dL	HCT, %	MCV, fL	% TIBC sat.	Ferritin, µg/L
12–15-yr-old girl	<11.8	<35.7	<81	<16	<18
12–15-yr-old boy	<12.5	<37.3	<82	<16	<15
>15-year-old girl	<12	<35.7	<85	<16	<15
>15-year-old boy	<13.3	<39.7	<85	<16	<15

Data from the Centers for Disease Control and Prevention. HCT = hematocrit; Hgb = hemoglobin; MCV = mean corpuscular volume; TIBC = total iron-binding capacity.

TABLE 7. Age-appropriate laboratory parameters to be monitored postadolescent obesity surgery

Nutritional factor	Laboratory parameter	Normal value	Effects of deficiency
Thiamin (B <sub>1</sub> )	Red blood cell transketolase stimulation	<15%	Beriberi, neuritis, edema, cardiac failure, anorexia, hoarseness, restlessness, aphonia
Pyridoxine (B <sub>6</sub> )	Plasma PLP	<20 nmol/L	Neuropathy, photosensitivity
Folic acid (B <sub>9</sub> )	Serum folate, RBC folate, homocysteine	>6 ng/mL, >160 ng/mL	Megaloblastic anemia, irritability, paranoid behavior
Cobalamin (B <sub>12</sub> )	Urine/serum B <sub>12</sub> *	<3.60 mmol/mol creatinine or 200–900 pg/mL	Pernicious anemia, neurological deterioration
Vitamin A	Plasma retinol	20–72 µg/dL	Night blindness, xerophthalmia, dermatomalacia, impaired resistance to infection, follicular hyperkeratosis, poor bone growth
Calcium	Ionized calcium	4.48–4.92 mg/dL	Numbness and tingling in the fingers, muscle cramps, convulsions, lethargy, poor appetite, and abnormal heart rhythms
Vitamin D	Serum 25-OHD	<50 nmol/L	Rickets, osteomalacia
Vitamin C	Plasma vitamin C	0.2–2 mg/dL	Bleeding gums, diarrhea, perifollicular hemorrhage, scurvy
Vitamin E	Plasma α-tocopherol	0.7–10 mg/dL	Hyporeflexia spinocerebellar and retinal degeneration
Zinc	Serum zinc	0.75–1.2 mg/L	Anorexia, hypogeusia, delayed growth or sexual maturation, impaired wound healing
Magnesium	Serum magnesium	1.5–2.0 mg/dL	Convulsions, neuropsychiatric disorders, hypomagnesemia
Copper	Serum copper	1.10–1.45 mg/L	Microcytic, hypochromic anemia, delayed growth osteoporosis, neutropenia

This table is a guide. Many laboratories have variability in the parameters that are checked as well as normal values. It is recommended that the clinician become familiar with the laboratory that typically serves their patients. If a deficiency is diagnosed, then it is recommended to repeat the nutrient as per traditional nutrient repletion guidelines (34,63,64). 25-OHD = 25-hydroxy vitamin D; PLP = pyridoxal-5-phosphate; RBC = red blood cell.

\* If borderline low level, confirm deficiency with serum methylmalonic acid may be check.

## Weight Regain

Understanding the factors that contribute to postsurgical weight regain may assist in a better understanding of how to prevent/minimize weight regain. The factors that may contribute to weight regain have yet to be determined, but these factors can vary from anatomical and physiological adaptations over time (66) to compliance issues (67), food urges (68), abnormal eating habits (69), to severely decreased postoperative well-being (68). More recently, a study showed that decreased resting metabolic rate correlated with those individuals who had weight regain (70). The clinician may want to consider encouraging self-monitoring and assisting the adolescent with control over food urges as well as reinforcing the need for follow-up, all of which have been shown to correlate with decreased postsurgical weight regain.

## Academic Environment

Adolescent patients present issues related to the academic environment that adult WLS patients do not encounter. For adolescents in middle or high school, the clinician should advise them to carry a water bottle to sip on fluids throughout the day, request lunch be between 11 AM and 1 PM, possibly allow for a snack or protein supplement during class time, and preferably pack lunch to provide the healthiest options. A letter to the school nurse or administrator may be necessary to communicate the special needs of the WLS population.

Adolescents attending college on campus should plan ahead to follow the recommended portion controlled eating pattern. Using campus services such as the food service director or college nutritionist can help them to navigate the system at their school. Most dining halls/eating plans at college provide a variety of nutritious options, with lean protein sources readily available. Many campuses offer a program through which food can be purchased by the ounce, cup, or half portion. Students should plan the timing and frequency of their meals that realistically accommodates their class schedule. Keeping their dorm room stocked with protein-rich foods, whole-grain snacks, water, and sugar-free drinks will allow them to make healthy, appropriate choices and minimize unplanned snacking on high-sugar, high-fat foods that provide much energy but few nutrients.

## Reproductive Health

The American College of Obstetrics and Gynecology recommends avoiding pregnancy for 12 to 18 months post-WLS (71). Little is known about the safety of a developing fetus in the setting of rapid weight loss and energy and nutritional restriction that occurs after WLS. Nutritional deficiencies are another concern for any patient having had a malabsorptive procedure that would place her at increased risk of folic acid and vitamin B<sub>12</sub> deficiency, which in turn increases the risk of an open neural tube defect. To date, there have been 7 infants with neural tube defects ranging from spina bifida to anencephaly born to mothers more than 12 months after gastric bypass surgery (72–74). Considering that there is

documentation of the lack of long-term noncompliance with taking vitamin supplements as directed, special attention should be focused on girls after WLS, especially those who have had malabsorptive procedures (75,76). Counseling should include the risk to the fetus if vitamins are not used in the first few months of pregnancy, often when a girl is unaware she is yet pregnant. For the majority of girls beyond the 12 to 18 months after WLS, there is good evidence that mothers and infants have no increased risk of complications related to prior WLS aside from biliopancreatic diversion (77). Additionally, because of the dramatic improvement in comorbidities such as hypertension, type 2 diabetes mellitus, and polycystic ovary syndrome after bariatric surgery, many experts argue that surgery may be beneficial in terms of reproductive health outcomes, in addition to the benefits already documented.

WLS programs should include either counseling regarding reproductive health concerns and recommendations as part of their pre- and postoperative assessments or referral to someone who is familiar with the commonly observed reproductive health problems among severely obese girls. These concerns include anovulatory menstrual cycles, polycystic ovary syndrome, increased risk of endometrial hyperplasia, increased infertility, and concerns related to gestational and type 2 diabetes mellitus. Additionally, there should be direct instruction regarding the recommendation to avoid pregnancy in the 12 to 18 months after surgery. Counseling regarding appropriate methods to prevent pregnancy should be provided to any sexually active girl planning WLS. Postoperatively, providers should continue to monitor for vitamin deficiencies, paying special attention to folic acid, vitamin B<sub>12</sub>, and iron replacement for girls desiring to become pregnant. Continuation of appropriate dietary intake in the WLS patient along with vitamin and mineral supplementation is important after delivery to ensure appropriate growth and development of the breast-fed infant (77,78). Inadequate weight gain reversed with the use of formula has been reported in a breast-fed infant born postgastric bypass with inadequate protein intake (79). Congenital vitamin B<sub>12</sub> deficiency with pancytopenia, global developmental delay, and cortical atrophy has been reported in breast-fed infants born to mothers several years after gastric bypass surgery (80,81). Consultation with a pediatric dietician and lactation consult during infancy can help ensure appropriate growth and development for the infant.

## Predictors of Success

Success after bariatric procedures requires significant behavioral changes that are dependent on the patient's ability to successfully implement lifestyle changes (82). Because of concerns about the ability to comprehend and implement longitudinal changes after bariatric surgery, many adult centers exclude patients with significant cognitive impairment. A recent survey of bariatric programs conducted by Bauchowitz et al (83), reported that 81.5% of the respondents would exclude individuals with an IQ <50 from consideration for bariatric procedures. There is a paucity of published data on bariatric procedures in adolescents with cognitive impairment, aside from patients with Prader-Willi syndrome (PWS), an imprinting disorder arising from the loss of the paternal copy of 15q11.2–13 with impairment in satiety and propensity to severe obesity as primary features. Bariatric procedures in children with PWS should be considered with extreme caution. A recent review of bariatric surgical outcomes in individuals with PWS noted poor results in comparison to nonsyndromic obese individuals (84).

The goal of bariatric surgical procedure is weight loss. In neither restrictive procedures, such as lap band, nor restrictive-malabsorptive procedures, such as RYGB, does weight loss lead to a

person reaching his or her ideal body weight. Often a percentage of excess weight (% EWL), the amount of excess weight that a patient has relative to his or her ideal weight, is the measure used to describe the success after a bariatric procedure. For bariatric surgery % EWL ranges from 58% to 64%. Restrictive procedures such as lap band have less % EWL (40%–54%), whereas the restrictive-malabsorptive procedures such as RYGB have greater mean % EWL, 56% to 74% (85). Change in BMI is also used to describe weight loss after a bariatric procedure. BMI is defined as the individual's body weight divided by the square of his or her height. Changes in BMI have also been used to define successful bariatric procedure. Presently there are good standards correlating BMI to cardiovascular risk factors. Finally, resolution or improvement with obesity-related comorbidities is another method of defining successful bariatric procedure. Resolution can be seen in type 2 diabetes mellitus, hyperlipidemia, hypertension, and obstructive sleep apnea.

Although there is a lack of data on predictive factors of success for pediatric patients undergoing bariatric surgery, extrapolation from the adult data would indicate the best chance for success surgery, defined as EWL >50% in individuals younger than 40 years with initial BMI <50 kg/m<sup>2</sup>, willingness to change eating habits, and willingness to increase physical activity (85). Although adult patients with extreme obesity do not seem to have good EWL, in adolescent patients this may not hold true. Data also exist highlighting the importance of the experience of the surgical team in conjunction with a multidisciplinary approach to weight loss, including behavior modification as important factors for optimal outcomes (86).

## CONCLUSIONS

Careful considerations should be made when assessing, educating, and monitoring the adolescent WLS patient. Most of these recommendations are based on the best practices of those currently working with this population. The recommendations in this article are meant to provide a guide when developing a new program or as a tool to reevaluate an existing program. At this point, there is limited evidence-based practice. Because of this, many WLS programs often vary in their approach, diet progression, and the like. It is anticipated that there will be more research in this area, and this assists in establishing more uniform protocols in WLS programs.

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# Maternal Obesity, Gestational Weight Gain, and Offspring Adiposity: The Exploring Perinatal Outcomes among Children Study

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**Objective** To determine whether adequate vs excessive gestational weight gain (GWG) attenuated the association between maternal obesity and offspring outcomes.

**Study design** Data from 313 mother-child pairs participating in the Exploring Perinatal Outcomes among Children study were used to test this hypothesis. Maternal prepregnancy body mass index (BMI) and weight measures throughout pregnancy were abstracted from electronic medical records. GWG was categorized according to the 2009 Institute of Medicine criteria as adequate or excessive. Offspring outcomes were obtained at a research visit (average age 10.4 years) and included BMI, waist circumference (WC), subcutaneous adipose tissue (SAT) and visceral adipose tissue, high-density lipoprotein cholesterol, and triglyceride levels.

**Results** More overweight/obese mothers exceeded the Institute of Medicine GWG recommendations (68%) compared with normal-weight women (50%) ( $P < .01$ ). Maternal prepregnancy BMI was associated with worse childhood outcomes, particularly among offspring of mothers with excessive GWG (increased BMI [20.34 vs 17.80 kg/m<sup>2</sup>], WC [69.23 vs 62.83 cm], SAT [149.30 vs 90.47 cm<sup>2</sup>], visceral adipose tissue [24.11 vs 17.55 cm<sup>2</sup>], and homeostatic model assessment [52.52 vs 36.69], all  $P < .001$ ). The effect of maternal prepregnancy BMI on several childhood outcomes was attenuated for offspring of mothers with adequate vs excessive GWG ( $P < .05$  for the interaction between maternal BMI and GWG status on childhood BMI, WC, SAT, and high-density lipoprotein cholesterol).

**Conclusion** Our findings lend support for pregnancy interventions aiming at controlling GWG to prevent childhood obesity. (*J Pediatr* 2014; ■: ■-■).

The prevalence of obesity has been increasing dramatically in the US, including among women of reproductive age.<sup>1</sup> Maternal obesity is a major risk factor for gestational diabetes mellitus (GDM) and future type 2 diabetes.<sup>2</sup> Moreover, observational studies suggest an independent association of maternal obesity with excessive fetal growth<sup>3,4</sup> and childhood obesity.<sup>5</sup> Alarming, increasing obesity trends are now observed early in life, even among young infants,<sup>6</sup> pointing toward harmful changes in the environment in which contemporary children are born and raised.<sup>7</sup> These and other observations lead to the hypothesis that maternal obesity during pregnancy is associated with lifelong consequences in the offspring<sup>8</sup> and, possibly, over successive generations.<sup>9</sup> It has been suggested that a transgenerational “vicious cycle” results, explaining at least in part, the increases in obesity, GDM, and type 2 diabetes seen over the past several decades.<sup>10</sup> In addition, obese children tend to become obese adults and, once present, obesity and its consequences are expensive and difficult to treat. This makes pregnancy a crucial window of opportunity for obesity prevention in this and the next generation.

The role of gestational weight gain (GWG) on childhood adiposity outcomes is less clear and incompletely studied. Some, but not all,<sup>11,12</sup> epidemiologic studies have found that greater GWG is associated with greater body mass index (BMI) in childhood<sup>13-17</sup> and adolescence<sup>5</sup> and with increased fat mass and poorer metabolic and vascular traits at age 9 years.<sup>18</sup> Some studies have suggested that the association of greater maternal weight gain and offspring obesity persists into adulthood.<sup>12</sup> Maternal prepregnancy BMI and excessive GWG have been linked independently to increased adiposity in the offspring.<sup>11,16,17,19-21</sup> In a group of preschool children, the odds of being categorized as overweight by age 4-5 years was increased by 57% in children exposed to both a maternal prepregnancy BMI greater  $\geq 25$  kg/m<sup>2</sup> and excessive weight gain during pregnancy.<sup>16</sup> It remains unclear however, whether the effect of maternal prepregnancy BMI on childhood adiposity outcomes is mitigated by adequate weight gain during pregnancy.

BMI	Body mass index	IOM	Institute of Medicine
EPOCH	Exploring Perinatal Outcomes among Children	KPCO	Kaiser Permanente of Colorado Health Plan
GDM	Gestational diabetes mellitus	SAT	Subcutaneous adipose tissue
GWG	Gestational weight gain	TG	Triglyceride
HDL-C	High-density lipoprotein cholesterol	VAT	Visceral adipose tissue
HOMA-IR	Homeostatic model assessment	WC	Waist circumference

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The answer to this question is important because it would provide support to the notion that healthier weight-gain patterns during pregnancy may improve the short- and long-term effects on offspring who have been exposed to maternal obesity. To address this question, we used data from the Exploring Perinatal Outcomes among Children (EPOCH) study in Colorado.

## Methods

The EPOCH study is an observational historical prospective cohort comprising children born between 1992 and 2002 at a single hospital in Colorado, whose biological mothers were members of the Kaiser Permanente of Colorado Health Plan (KPCO) and who were offspring of singleton pregnancies. All children exposed to maternal GDM were eligible, together with a random sample of children not exposed to GDM. Children were invited to attend an in-person research visit when they were on average age 10.5 years (range, 6-13 years), and approximately 68% agreed to participate.

Included in this analysis were 313 mother-child pairs (141 non-Hispanic white, 145 Hispanic, 27 non-Hispanic African American) who were part of the EPOCH study and had complete data on maternal prepregnancy BMI, GWG, and offspring adiposity outcomes. Children and their mothers completed a research visit between January 2006 and October 2009. Because the EPOCH study was specifically designed to explore the long-term effects of maternal GDM on offspring, the cohort is enriched in offspring of GDM mothers. Because we were exploring specific hypotheses regarding the role of excessive GWG as effect modifier, the small number of offspring of mothers who gained insufficient gestational weight during pregnancy was excluded. The study was approved by the local Institutional Review Board, and all participants provided written informed consent and youth provided written assent.

### Maternal Measures

Maternal pregnancy measures (weight, GDM) and offspring birth weight were obtained from the KPCO perinatal database, a linkage of the maternal and perinatal medical record containing prenatal and delivery events for each woman. Maternal prepregnancy weight was measured before the last menstrual cycle preceding pregnancy. Maternal height was collected at the in-person research visit and used to calculate prepregnancy BMI ( $\text{kg}/\text{m}^2$ ). BMI was categorized as normal weight ( $18.5\text{-}25 \text{ kg}/\text{m}^2$ ) and overweight/obese ( $\geq 25 \text{ kg}/\text{m}^2$ ). Multiple weight measures during pregnancy (on average 4 per participant) were used to model GWG using a longitudinal mixed effects model that included fixed effects for time, time squared, prepregnant BMI, maternal age, gravidity, and a time by prepregnant BMI interaction. The model included subject-specific intercept and slope terms. GWG was estimated using the absolute predicted weight gain for a full-term pregnancy (model predicted weight at term minus model predicted weight at conception). Women were categorized as either exceeding or meeting the 2009 recommended Institute of Medicine (IOM) GWG guidelines (adequate total

GWG for normal BMI prepregnancy  $11.4\text{-}15.9 \text{ kg}$  and overweight/obese BMI prepregnancy  $5\text{-}11.4 \text{ kg}$ ).<sup>22</sup> Women who gained inadequate weight during pregnancy were excluded from this analysis, according to our a priori hypothesis.

Physician-diagnosed GDM was coded as present if diagnosed through the standard KPCO screening protocol and absent if screening was negative. Since the 1990s, KPCO has routinely screened for GDM in all nondiabetic pregnancies using a 2-step standard protocol and criteria based on the National Diabetes Data Group recommendations.<sup>23</sup>

### Childhood Measures

All children were invited to an in-person research visit, which included anthropometric measures, questionnaires, a magnetic resonance imaging exam of the abdominal region and a fasting blood sample. Race/ethnicity was self-reported using 2000 US census-based questions and categorized as Hispanic (any race), non-Hispanic white, or non-Hispanic African American. Pubertal development was assessed by child self-report with a diagrammatic representation of Tanner staging adapted from Marshall and Tanner.<sup>24</sup> Youth were categorized as Tanner  $<2$  (prepubertal) and  $\geq 2$  (pubertal). Total energy intake (kilocalories per day) was assessed using the Block Kid's Food Questionnaire.<sup>25</sup> Self-reported key activities, both sedentary and nonsedentary, performed during the previous 3 days were measured using a 3-day Physical Activity Recall questionnaire.<sup>26</sup> Each 30-minute block of activity was assigned a metabolic equivalent variable to accommodate the energy expenditure. Results were reported as the average number of 30-minute blocks of moderate-to-vigorous activity per day. Current height and weight were measured in light clothing and without shoes. Weight was measured to the nearest 0.1 kg using a portable electronic SECA scale (SECA, Chino, California). Height was measured to the nearest 0.1 cm using a portable SECA stadiometer. Height and weight were measured and recorded twice, and an average was taken. Scales and stadiometers were calibrated every 2 months using standard weights for scales and an aluminum measuring rod for the stadiometer. BMI was calculated as  $\text{kg}/\text{m}^2$ . Waist circumference (WC) was measured to the nearest 1 mm at the midpoint between the lower ribs and the pelvic bone with a metal or fiberglass nonspring-loaded tape measure.

Magnetic resonance imaging of the abdominal region was used to quantify visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) with a 3T HDx imager (General Electric, Waukesha, Wisconsin) by a trained technician. Each participant was placed supine and a series of T1-weighted coronal images were taken to locate the L4/L5 plane. One axial, 10-mm, T1-weighted images at the umbilicus or L4/L5 vertebra was analyzed to determine SAT and VAT content. The analysis technique used was a modification of the technique of Engelson,<sup>27</sup> where adipose tissue regions were differentiated by their signal intensity and location. Images were analyzed by a single reader.

Cholesterol, triglyceride (TG), and high-density lipoprotein cholesterol (HDL-C) were obtained while the patient

was fasting and measured using the Olympus (Center Valley, Pennsylvania) AU400 advanced chemistry analyzer system. Estimated insulin resistance was based on the homeostatic model assessment (HOMA-IR), calculated using fasting glucose and insulin levels collected at the study visit according to the formula: (fasting glucose [mmol/L]  $\times$  fasting insulin [ $\mu$ U/mL]/22.5).

### Statistical Analyses

All analyses were conducted using SAS v9.4 (SAS Institute, Cary, North Carolina). Prepregnant BMI was categorized as normal weight (18.5-25 kg/m<sup>2</sup>) and overweight/obese ( $\geq$ 25 kg/m<sup>2</sup>). Descriptive analyses compared prepregnant BMI groups using both *t* tests and  $\chi^2$  tests (Table I). Univariate regression was used to examine whether maternal prepregnant BMI was associated with childhood adiposity-related variables (including BMI, WC, SAT and VAT, HDL-C, TGs, and HOMA-IR). An ANOVA model was used to investigate the modifying effect of GWG on the relationship between prepregnancy maternal BMI and childhood adiposity-related variables. All models were controlled for potential confounders, which included current offspring age, sex, race/ethnicity, Tanner stage, birth weight, and maternal GDM status.

## Results

Table I shows the characteristics of the study population, according to maternal prepregnant obesity status. Of the 313

eligible mothers, 164 were classified as overweight or obese with a prepregnant BMI  $>$ 25 kg/m<sup>2</sup>. The groups were similar in terms of maternal age, parity status, and gestational age at delivery. Women categorized as overweight or obese were more likely to be diagnosed with GDM compared with normal-weight women, although not significantly ( $P = .09$ ). Maternal GWG patterns according to maternal prepregnancy overweight/obesity status are also described in Table I. As expected, normal-weight mothers gained significantly more weight in each trimester of pregnancy and overall, compared with overweight/obese mothers. However, 68% of overweight/obese mothers exceeded the 2009 GWG IOM recommendations vs only 50% of normal weight mothers ( $P < .01$ ).

Children in the 2 groups were similar in terms of age, sex, and pubertal status, but significantly more offspring of overweight/obese mothers were of Hispanic or African American descent ( $P = .004$ ).

All childhood adiposity measurements were significantly different according to maternal prepregnancy obesity status (Table I). Offspring of overweight/obese mothers had significantly greater BMI, WC, as well as subcutaneous and visceral fat, compared with offspring of normal-weight mothers. TGs and insulin resistance, as estimated by risk HOMA-IR, also were significantly worse for the children exposed to overweight/obesity in utero.

Table II shows the association between maternal prepregnancy BMI and various offspring adiposity-related outcomes. The analysis is stratified by whether the mothers

**Table I.** Characteristics of study population (mean  $\pm$  SD) according to maternal prepregnancy overweight/obesity status

	Normal weight (n = 149)	Overweight or obese (n = 164)	P value
<b>Maternal characteristics</b>			
Maternal age at delivery, y	30.38 $\pm$ 5.73	30.66 $\pm$ 5.84	.67
Nulliparous, n (% yes)	42 (28.19)	50 (30.49)	.66
Prepregnant BMI, kg/m <sup>2</sup>	21.70 $\pm$ 1.99	30.76 $\pm$ 5.29	<.0001
GDM, n (%)	24 (16.11)	39 (23.78)	.09
First-trimester weight gain, kg	3.98 $\pm$ 1.07	3.18 $\pm$ 1.51	<.0001
Second-trimester weight gain, kg	4.88 $\pm$ 1.08	4.08 $\pm$ 1.53	<.0001
Third-trimester weight gain, kg	5.68 $\pm$ 1.07	4.89 $\pm$ 1.51	<.0001
Total GWG, kg	16.52 $\pm$ 3.55	13.58 $\pm$ 5.41	<.0001
Exceeded IOM recommended GWG, n (%)	74 (49.66)	111 (67.68)	.0012
<b>Child characteristics</b>			
Gestational age at delivery, wk	38.86 $\pm$ 1.79	38.74 $\pm$ 2.43	.62
Birth weight, g	3211.30 $\pm$ 551.00	3250.10 $\pm$ 653.30	.57
Current age, y	10.48 $\pm$ 1.41	10.39 $\pm$ 1.50	.59
Male sex, n (%)	81 (54.36)	91 (55.49)	.84
Race/ethnicity, n (%)			.004
Non-Hispanic white	80 (53.69)	61 (37.20)	
Hispanic	62 (41.61)	83 (50.61)	
Non-Hispanic African American	7 (4.70)	20 (12.20)	
Tanner stage $\geq$ 2, n (%)	77 (46.95)	87 (53.05)	.25
BMI, kg/m <sup>2</sup>	17.80 $\pm$ 3.31	20.34 $\pm$ 5.14	<.0001
Waist, cm	62.83 $\pm$ 9.02	69.23 $\pm$ 13.50	<.0001
SAT, cm <sup>2</sup>	90.47 $\pm$ 78.05	149.30 $\pm$ 119.50	<.0001
VAT, cm <sup>2</sup>	17.55 $\pm$ 9.98	24.11 $\pm$ 15.80	<.0001
TG, mg/dL	82.72 $\pm$ 37.91	93.51 $\pm$ 43.80	.02
HDL-C, mg/dL	52.72 $\pm$ 9.44	50.46 $\pm$ 11.14	.06
HOMA-IR	36.69 $\pm$ 26.92	52.52 $\pm$ 39.95	<.0001
Physical activity, blocks/day	4.52 $\pm$ 2.93	3.88 $\pm$ 2.88	.05
Total daily calories, kcal/d, mean (95% CI)	1795.60 $\pm$ 549.60	1839.40 $\pm$ 531.30	.47
Percent calories from fat, %	35.99 $\pm$ 5.11	36.20 $\pm$ 4.80	.70

**Table II.** The association between maternal prepregnancy BMI and offspring outcomes, stratified by GWG status\*

	Adequate GWG, $\beta$ (95% CI)	Excessive GWG, $\beta$ (95% CI)	Interaction, <i>P</i> value
BMI, kg/m <sup>2</sup>	0.13 (0.02-0.253)	0.34 (0.25-0.44)	.004
Waist, cm	0.38 (0.10-0.65)	0.83 (0.58-1.08)	.01
SAT, cm <sup>2</sup>	3.49 (0.89-6.08)	7.26 (4.90-9.62)	.03
VAT, cm <sup>2</sup>	0.37 (0.004-0.74)	0.72 (0.39, 1.06)	.16
TG, mg/dL	0.16 (−0.98, 1.31)	1.12 (0.08, 2.16)	.21
HDL-C, mg/dL	0.16 (−0.12, 0.44)	−0.34 (−0.60, −0.07)	.01
HOMA-IR	0.55 (−0.33, 1.43)	1.66 (0.86, 2.45)	.06

$\beta$  = the unit increase in offspring parameter for every 1 kg/m<sup>2</sup> increase in maternal prepregnancy BMI.

*P* values for each univariate model reported.

\*Adjusted for age, sex, race/ethnicity, Tanner stage, birth weight, and maternal GDM.

were meeting or exceeding the 2009 IOM recommendations for GWG. Increasing maternal prepregnancy BMI was associated with significantly worse childhood outcomes in both GWG groups. However, the effect of maternal prepregnancy BMI on several childhood outcomes was attenuated for offspring of mothers with adequate vs excessive GWG. This trend was observed for all explored variables and was statistically significant for childhood BMI, WC, SAT, and HDL-C. Additional adjustment for current diet and physical activity did not materially change the results.

The **Figure** illustrates the relationship between maternal prepregnancy BMI and childhood adiposity-related outcomes and the effect of adequate vs excessive GWG on this relationship. In each panel, the association of prepregnancy BMI and child outcome (BMI, WC, SAT, and HDL-C) is attenuated for offspring of mothers who gained adequate vs those who gain excessive gestational weight.

## Discussion

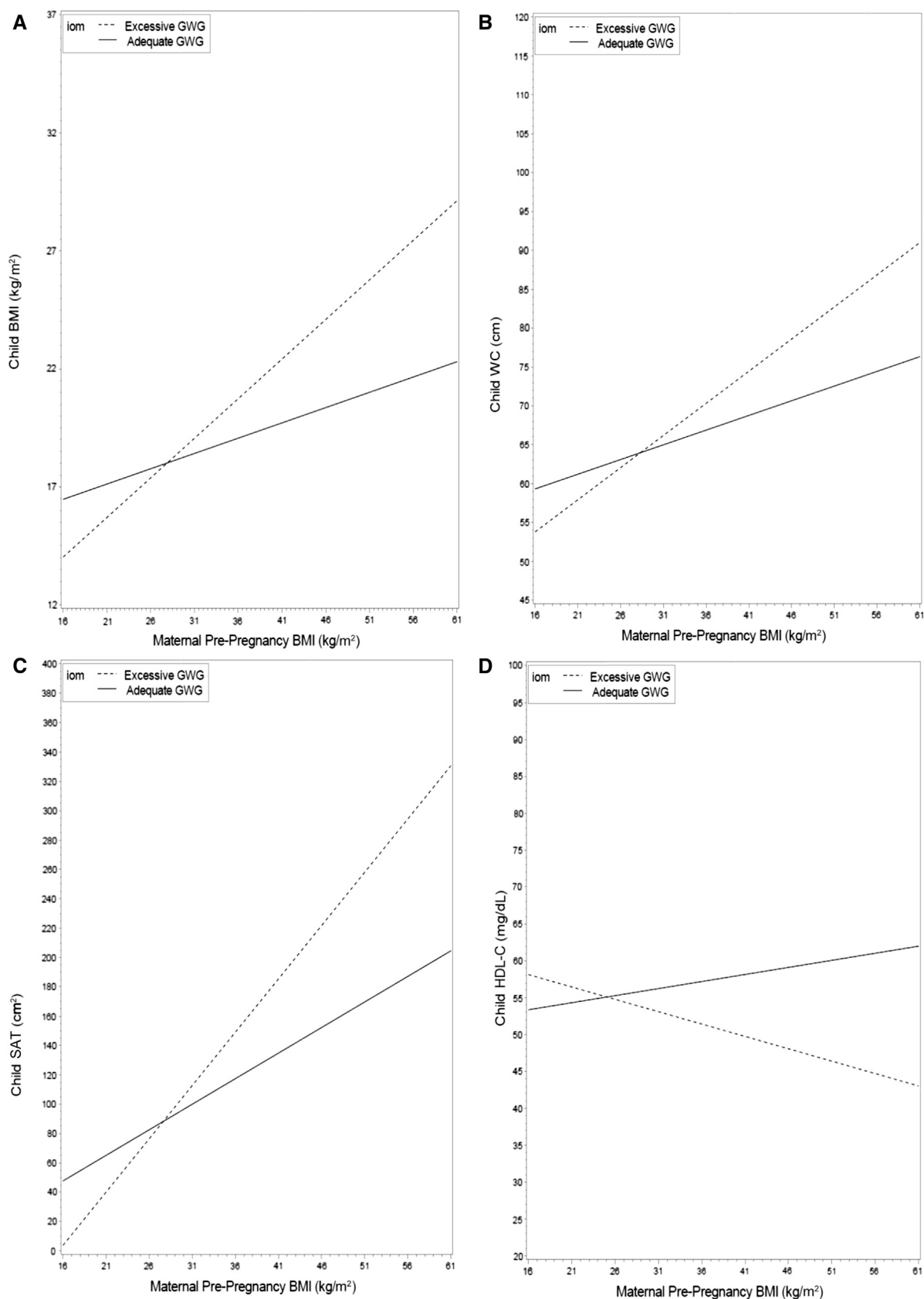
In this cohort of more than 300 mother-child pairs, women who were overweight or obese before their pregnancy were more likely to exceed the IOM recommendations compared with women who began their pregnancy at a normal BMI. Furthermore, greater prepregnancy BMI was associated with worse adiposity and metabolic risk markers in their offspring at an average age of 10 years, including greater BMI and WC, SAT and VAT fat deposition, and abnormal lipid markers. Several of these relationships were significantly attenuated, however, for offspring of women that gain the recommended amount of weight, compared with those who gained excessive weight during pregnancy.

This analysis extends previous observations of an association between maternal prepregnancy BMI and offspring adiposity outcomes later in life.<sup>11,20,21,28,29</sup> A clear relationship between maternal weight status before pregnancy has been linked to offspring obesity as early as 2-4 years of age in a retrospective cohort of low-income Women, Infants,

and Children participants,<sup>11</sup> in a national representative cross-sectional sample of 6-8-year-olds<sup>20</sup> and extended to early adolescence period in a prospective sample of more than 200 white mother-child pairs.<sup>21</sup> Various adiposity indicators in children have been explored, including BMI percentile<sup>11,20</sup> and fat mass via dual X-ray absorptiometry measurements.<sup>21</sup> Offspring of mothers who had a BMI  $\geq 30$  kg/m<sup>2</sup> prepregnancy had a greater odds of being categorized as obese ( $\geq 95$ th percentile) at age 2 years (OR 2.2 [95% CI 1.8-2.6]), 3 years (OR 2.6 [95% CI 2.2-3.1]), and 4 years (OR 2.6 [95% CI 2.2-3.1]).<sup>11</sup> Similarly, for every one-unit increase in maternal pregnancy BMI, fat mass measured by dual X-ray absorptiometry increased by 0.26 (95% CI 0.04-0.48) in boys and 0.42 (95% CI 0.29-0.56) in girls at age 9 years.<sup>21</sup>

Several mechanisms that are not mutually exclusive may explain these associations. These include shared genes, shared familial socioeconomic and lifestyle factors, as well as specific intrauterine effects. Work, particularly from the Pima Indian population, suggests that the effect of maternal pregnancy diabetes on offspring obesity risk is not fully explained by genetic factors. In a small nuclear family study (52 families, 182 siblings) conducted in the Pima Indian population, obesity was greater among nondiabetic offspring born after the mother had been diagnosed with type 2 diabetes (ie, overnutrition resulting from exposure to increased intrauterine glucose levels) than in their siblings born before their mothers' diagnosis (ie, exposed to lower intrauterine glucose levels).<sup>30</sup> In another study the prevalence of obesity among 2- to 18-year-old siblings born after maternal biliopancreatic surgery was 52% lower than among age-matched siblings born when their mother was obese.<sup>31</sup> Because siblings discordant for intrauterine exposures carry a similar risk of inheriting the same susceptibility genes and share a similar postnatal environment, such studies provide strong evidence that part of the excess risk of childhood obesity associated with overnutrition in utero reflects specific intrauterine effects.

From a public health prevention perspective, distinguishing between specific intrauterine mechanisms and shared familial genetic/behavioral effects is essential for the development of randomized trials aimed at testing effective pregnancy interventions to reverse the obesity epidemic. In the absence of definite evidence provided by a randomized clinical trial, this question can be tested by exploring whether GWG is a potential effect modifier of the relationship between maternal BMI and child outcomes. We found that adequate GWG significantly reduces the association between maternal prepregnancy BMI and offspring outcomes. For most childhood adiposity-related outcomes, the association with maternal prepregnancy BMI was still significant even if mothers gained the recommended amount of weight during pregnancy, likely reflecting the other causal pathways described previously (shared familial genetic and nongenetic effects); however, all these associations were substantially reduced (by 50%-60%) if women gained the recommended amount of weight during pregnancy. Of note, the inverse association between maternal BMI and offspring HDL-C levels



**Figure. A-D,** GWG modifies the association between maternal prepregnancy BMI and childhood adiposity-related measurements.

observed with excessive weight gain during pregnancy became nonsignificant among the group who met the GWG recommendations.

Our study had numerous strengths, including directly measured pregnancy exposures, state-of-the-art measures of childhood adiposity, and the ability to readily explore associations between pregnancy exposures and childhood adiposity outcomes later in life. Limitations include the observational (rather than experimental) nature of the study and, likely, the relatively limited size of the cohort, which may have resulted in some nonsignificant interactions. We were underpowered to additionally explore whether insufficient GWG modifies the association between maternal BMI and offspring outcomes. However, the majority of research in this area has found no or little association between inadequate GWG and childhood risk of obesity.<sup>32</sup> Finally, our cohort has oversampled women with GDM and thus our findings may not be completely generalizable to a lower-risk population.

In conclusion, our findings suggest that the effect of maternal prepregnancy BMI on several childhood adiposity-related outcomes is attenuated for offspring of mothers with adequate vs excessive GWG. Therefore, pregnant women should be encouraged to follow the IOM recommendations of weight gain for their given prepregnancy BMI. Finally, our study lends support for pregnancy interventions aiming at controlling GWG to prevent childhood obesity. Carefully designed randomized clinical trials are needed to determine whether improved weight gain patterns can be achieved throughout pregnancy that would prevent the short and long-term consequences on the offspring, and curb the obesity epidemic. ■

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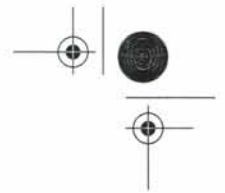
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# 7 Clinical Evaluation: Diagnosis, Medical Testing, and Follow-up

*Nancy F. Krebs and Melinda S. Sothorn*

## CONTENTS

Prevalence.....	80
Causes/Risk Factors .....	80
Comorbidities .....	81
Treatment Approaches .....	81
Diagnosis .....	82
Medical History .....	82
Family History .....	83
Social History .....	83
Review of Systems .....	84
Primary Risk Factors .....	84
Physical Exam .....	84
Body Mass Index.....	85
Background.....	85
Calculating Obesity Risk and Status with BMI Percentiles.....	85
Diagnosis .....	85
Anthropometrics to Assess Fat Distribution .....	85
Skinfolds .....	85
Waist Circumference .....	85
Direct Measurements of Body Composition .....	86
Laboratory Testing — Basic Panels.....	86
Measurements of Energy Expenditure.....	86
Referrals .....	86
Genetics .....	86
Endocrinology.....	86
Thyroid .....	88
Pancreas .....	88
Cardiology .....	88
Pulmonary .....	88
Orthopedics and Physical Therapy .....	89
Psychology.....	89
Social Services.....	89
Nutrition.....	89
Exercise.....	89



Education.....	89
Follow-Up.....	90
At Risk for Obesity.....	90
Overweight.....	91
Severe Overweight.....	91
Summary.....	91
References.....	92

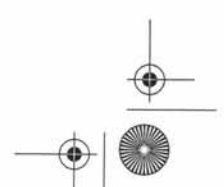
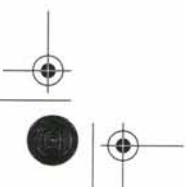
## PREVALENCE

The rapid rise in the prevalence of childhood overweight and obesity is occurring in both industrialized and developing countries all over the world. Pediatricians and other pediatric health care providers will play an increasingly important role in the early identification and prevention of childhood obesity and its associated comorbid conditions [1,2]. Because obesity is usually established at a young age, the pediatric primary care office is critical to national efforts to reverse the pediatric obesity epidemic [3]. According to the 1999–2000 National Health and Nutrition Examination Survey, the prevalence of childhood obesity (at or above the 95th percentile for body mass index [BMI] on standard reference growth charts) in the United States has grown to 15.3% of 6 to 11 year olds and 15.5% of 12 to 19 year olds [2,4]. Obesity prevalence has been found to be even higher among minority and economically disadvantaged populations [2,4,5].

## CAUSES/RISK FACTORS

Lower levels of daily physical activity by children in the United States have lead to a greater number of health problems in children than in previous generations. Sedentary lifestyles increase the risk of childhood medical conditions such as obesity, hypertension, hyperinsulinemia, hypercholesterolemia, and dyslipidemia [2,6]. Studies show that parent inactivity strongly predicts child inactivity [7,8]. A recent study examined the self-reported physical activity and dietary intake patterns of parents and changes in weight status over 2 years in offspring [9]. Girls of parents with high dietary intake and low physical activity (obesogenic) had significantly greater increases in weight status. Thus, in addition to family history of obesity, the environment of the home may equally contribute to the risk for developing obesity in childhood. However, there are also strong arguments for the effect of the genetic profile and the early nutritional environment on the risk for developing obesity during childhood [10–15]. Jackson and colleagues provide a strong argument for nutrition-induced changes in the hypothalamic–pituitary–adrenal axis in the mother and the fetus [10]. It is suggested that the local availability of nutrients during pregnancy, especially in relation to protein intake, may negatively affect future metabolic health. Adjustments may occur to protect brain tissue preferentially over visceral and somatic growth, resulting in an altered metabolic profile [10]. Thus, nutrition during pregnancy may have strong implications for future obesity and related chronic disease.

Infancy is also considered a critical period for obesity development. A high protein intake at the age of 2 years was shown to promote increased fatness at 8 years of age, suggesting that a high-protein diet early in life could promote an increased risk of obesity later in childhood, but findings in this area are limited and have not been consistent [16]. Moreover, research generally supports that children who were breastfed have a lower risk of obesity than those who were formula-fed [17–20]. In addition, those infants who breastfed for longer durations showed an even lower risk of childhood obesity [21]. Differences in feeding between breastfed and formula fed infants may also have a critical influence on infant weight gain. Therefore, low birth weight and breastfeeding history should be considered factors in obesity development in young children (Table 7.1). In addition, children with such risk factors may be predisposed genetically and behaviorally to the early manifestation of subtle, nonsymptomatic metabolic abnormalities that lead to childhood obesity and related chronic disease [22–28]. Therefore, strategies that positively alter the nutrition



**TABLE 7.1**  
**Risk Factors for Pediatric Obesity**

---

Socioeconomic status
Ethnicity
Parental obesity — under 6 years of age
Body mass index — over 6 years of age
Critical development periods
Birth (low/high birth weight)
5–6 years of age (adiposity rebound)
Puberty (12–15 years of age)
Infant formula feeding
Poor nutrition, food preferences
Excessive sedentary behaviors

---

and physical activity behaviors and environment of the family may reduce the risk of obesity in young children, especially in those with one or more risk factors. A recent publication of the American Academy of Pediatrics offers pediatric obesity prevention guidelines for medical professionals, which include increased monitoring of at-risk children and parent education [2].

Pediatric health care providers should recognize that environmental factors may greatly affect physical activity patterns. Unsafe neighborhoods and lack of adult supervision after school may increase time spent in sedentary behavior such as watching television and playing DVDs or video or computer games [29].

## COMORBIDITIES

Pediatric obesity is associated with many significant health problems and is strongly linked to increased risks for adult obesity, related comorbid diseases, and shortened life expectancy [2,6]. Growing numbers of obese children exhibit early signs of diseases that were once found only in adult populations including type 2 diabetes mellitus, high blood pressure, and abnormal lipid profiles [3]. Obese children are at an increased risk for diseases that can affect the cardiovascular, pulmonary, endocrine, and gastrointestinal systems, as well as orthopedic conditions and psychological health problems [2,6]. Comorbidities affecting the cardiovascular system include hypercholesterolemia, hypertension, and dyslipidemia [2,6a,30]. Endocrine system comorbidities include hyperinsulinemia, insulin resistance, impaired glucose tolerance, type 2 diabetes mellitus, and menstrual irregularity [2]. Common pulmonary and gastrointestinal comorbidities include sleep apnea, asthma, obesity hypoventilation syndrome, and nonalcoholic steatohepatitis [2,29]. Common orthopedic comorbidities include slipped capital femoral epiphysis, Blount disease (tibia vara), and genu varum. Mental health problems may include depression and low self-esteem [2,6,29].

## TREATMENT APPROACHES

Several weight loss approaches can be considered when choosing a treatment plan for an overweight or obese patient. Because obesity is a multifactorial disease, treatment of the disease is approached from many angles including diet modification, increased physical activity, psychological intervention, pharmacotherapy, and surgery. Reduction of energy intake is a mainstay of treatment, but the ideal approach will vary with the skills and motivation of the family, the severity of the overweight status, and the age of the child. Caution should be used in prescribing “diets” for children, except under well-supervised conditions because of the risks associated with overly restrictive access to food. This has been associated (in young children) with decreased ability to self-regulate energy intake [31,32]. In adolescents, dieting was inversely associated with BMI [33]. Several dietary

approaches can be considered including low fat, low carbohydrate, high protein, and low glycemic index. The Traffic Light Diet (see Appendix A3.3B), which applies principles of foods with high versus low caloric density, has been shown to be easy for children to understand and successfully follow [34]. Other popular treatments focus on psychological and family therapy, including behavioral modification [35]. Behavioral modification has been shown to increase the success of obesity treatment and includes such elements as goal setting, maintaining a food diary, reducing availability of high-calorie stimulus foods, positive reinforcement, and parental support [34]. Health providers should seek training in health behavior change techniques, parent limit-setting strategies and reinforcement skills, and family conflict awareness [36]. Dietary treatment should be accompanied by efforts to emphasize increased physical activity [34,35]. However, exercise alone is generally not sufficient to promote significant weight loss without diet modification [34]. Physical activity provides additional health benefits and is linked to successful maintenance of weight-loss. Reduction of sedentary activities such as television viewing, video games, and computer time may be especially effective targets for behavior change [6a,37,38].

Pharmacotherapy is sometimes a useful adjunct to diet, activity, and behavioral change strategies. Examples specific for obesity treatment include sibutramine and orlistat, but each has significant potential side effects, and efficacy data are limited. Bariatric surgery has been successfully performed on severely obese adolescents. Guidelines for patient and site selection have been published [39]. Criteria include a BMI of at least 40, accompanied by significant comorbidities such as obstructive sleep apnea, type 2 diabetes mellitus, and pseudotumor cerebri. This treatment is best undertaken in a center that has surgeons with experience with the procedure in adolescents, and at which a multidisciplinary team is available. The procedure appears to be safe in the short term, and weight loss is typically substantial, with improvement in comorbidities. There are currently few data on long-term outcomes and complications [34,35,40].

## DIAGNOSIS

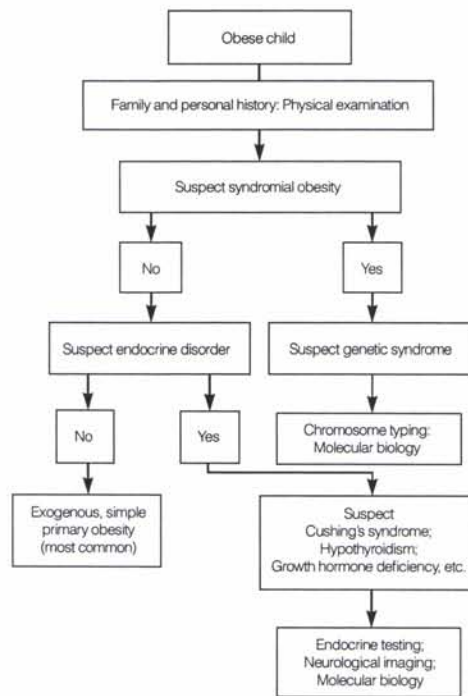
Early diagnosis and treatment of obesity in children is crucial for the successful management of pediatric health [2,41,42]. Unfortunately, although childhood obesity has now reached epidemic levels, this disease is still under-recognized by the health care community [43]. In addition, underdiagnosis is generally more prevalent than misclassification of obesity [44]. Recently, O'Brien et al. [43] reported that pediatric health care providers diagnosed overweight in only one-half (53%) of overweight children examined for health supervision. Moreover, in children diagnosed as overweight by their physicians, comprehensive treatment programs were not generally prescribed [43]. One study has shown that although plotting BMI enhanced physician recognition of overweight when compared with plotting height and weight for age, survey data in the same report indicated that only a minority of pediatricians routinely use BMI [45]. In Chapter 4 of this volume, Gordon provides similar disappointing rates of diagnosis and referral in overweight children in primary care settings [46]. Kiess and colleagues provide a diagnostic algorithm for childhood obesity that primary care providers can use to determine the most appropriate management plan (Figure 7.1) [35].

## MEDICAL HISTORY

Pediatric obesity is a complex condition that is associated, as noted above, with a plethora of medical complications, syndromes, and disorders. An expert panel report [47] suggests that the initial diagnosis should begin with a thorough screening of the patient's medical history. Conditions of primary concern are hypertension, endocrine disorders, orthopedic problems, type 2 diabetes mellitus (or insulin resistance), genetic syndromes, sleep disorders, pseudotumor cerebri, and gastrointestinal disorders.

As part of this medical history, information on the child's current eating and physical activity patterns should be obtained, with a particular focus on behaviors that can be targeted for change.

## Clinical Evaluation: Diagnosis, Medical Testing, and Follow-up



**FIGURE 7.1** Diagnostic algorithm for childhood obesity. Reprinted with permission from Kiess, W., A. Reich, G. Muller, A. Galler, T. Kapellen, K. Raile et al. Obesity in childhood and adolescence: clinical diagnosis and management. *J Pediatr Endocrinol Metabol.* 14(Suppl 6):1431–1440, 2001.

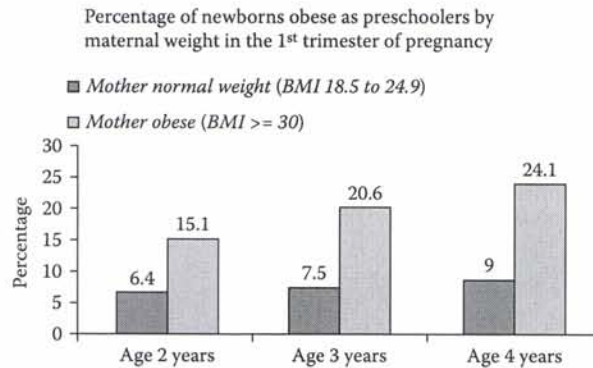
An expert committee has [29] emphasized that both food type and patterns of eating should be assessed to discern origins of excess caloric intake. Likewise, the assessment of physical activity patterns and extent of sedentary behaviors provides information important to increasing energy expenditure [29]. A comprehensive medical history and physical examination form may be found in Appendix A1.6.

### FAMILY HISTORY

Critical to assessment of a child's risk from overweight is the documentation of the family history of disease. The recommended conditions to evaluate when obtaining family history from the patient include overweight, dyslipidemia, hypertension, cardiovascular disease, gallbladder disease, eating disorders in parents, type 2 diabetes mellitus, and other endocrine abnormalities [47]. Emerging research indicates that the health status of the mother immediately before pregnancy, during pregnancy, and during breastfeeding may also be important [48]. Whitaker tracked the weight status of preschool children whose mothers were obese during pregnancy. These youth were twice as likely to be overweight as children whose mothers maintained a healthy weight during pregnancy (Figure 7.2) [49].

### SOCIAL HISTORY

The structure of the family, school and child care arrangements, living situation, parental employment, and history of abuse are examples of information to be obtained in the social history, which will inform the negotiations for health behavior changes.



**FIGURE 7.2** Percentage of newborns obese as preschoolers by maternal weight in the first trimester of pregnancy. Reprinted with permission from Whitaker, R. C. Predicting preschooler obesity at birth: the role of maternal obesity in early pregnancy. *Pediatrics*. 114:e29–36, 2004.

### REVIEW OF SYSTEMS

The review of systems should address symptoms of potential comorbidities of overweight. Examples include asking about the presence of headaches; visual changes; sleep problems such as snoring, restless sleep, inability to lie supine, and daytime somnolence; shortness of breath or wheezing; chest pain; abdominal pain; and joint and skeletal muscle complaints. A brief evaluation of the child's psychological status is also useful.

### PRIMARY RISK FACTORS

The children who are at the highest risk for becoming obese are those who belong to economically disadvantaged minority populations (Table 7.1). As a result of having a low socioeconomic status, children may have less access to safe places for physical activity and/or less access to healthful food choices such as fruits and vegetables. Recent studies show a consistent rise in the prevalence of obesity among preschool children from low-income families [50]. These children often have low levels of cognitive stimulation, which is associated with a significant increase in the risk for early-onset obesity [51]. Other risk factors that have been linked to an increased risk include unhealthy family and parental dynamics, low or high birth weight, maternal diabetes and obesity, high prevalence of obesity in other family members, and overcontrolling parental behavior (Table 7.1) [2].

### PHYSICAL EXAM

As for all patients, a patient who is found to be overweight should have a physical examination, in this case with a focus on signs of comorbid conditions that may be present or on any underlying conditions that may contribute to excessive weight gains, such as hypothyroidism, reactive airways disease, tonsillar hypertrophy (contributing to airway obstruction), genu varum (flat feet) or other orthopedic conditions, and genetic or endocrine abnormalities [41]. A patient with insulin resistance may show signs of acanthosis nigricans (darkening of the skin). Hypothyroidism should be suspected with excessive weight gain and plateauing of linear growth; exam findings may include skin and hair changes, enlarged thyroid, and absent deep tendon reflexes. An abdominal exam should assess liver size and tenderness. Postural and gait abnormalities may indicate the presence of orthopedic conditions such as genu varum. Tanner stage should be evaluated and assessed in relation to the child's age. Rare genetic and endocrine abnormalities may manifest themselves through dysmorphic features including abnormal genitalia, developmental delay, poor linear growth, hirsutism,

and striae [41]. Blood pressure measurements should also be obtained using an appropriately sized blood pressure cuff.

## BODY MASS INDEX

### Background

The World Health Organization, the Centers for Disease Control and Prevention, and many national organizations recommend the use of BMI to identify overweight and obesity in youth [2,52,53]. BMI is a convenient measurement for screening for overweight in children. Standard BMI classifications define a BMI between the 85th and 95th percentiles for age and sex as “at risk for overweight,” and a BMI greater than the 95th percentile for age as “overweight” (Appendix A1.10). BMI is an accepted screening tool for use by pediatric health care providers as a result of its use of easily accessible data (weight and height) and moderately strong correlation with laboratory measurements of body fatness [2,3,54].

### Calculating Obesity Risk and Status with BMI Percentiles

Pediatric growth charts for the U.S. population now include BMI percentile grids for age and gender and can be used for longitudinal tracking of a patient’s BMI from ages 2 through 20 years, and to identify overweight (see Appendix A1.10) [29,53]. BMI is calculated by applying one of the following formulas:

$$\text{weight (kilograms)/height (meters)}^2$$

or

$$[\text{weight (pounds)/height (inches)}^2] \times 703.$$

### Diagnosis

Once BMI is calculated, the physician or health care provider can determine risk and status by plotting on Centers for Disease Control and Prevention growth charts (see Appendix A1.10). This should be done at least annually to facilitate the early recognition of overweight and to monitor weight increases relative to linear growth [2]. If a trend for excessive weight gain is established (e.g., crossing BMI percentile channels or an increase of three to four BMI units in 1 year), contributing factors should be explored and discussed with parents to prevent further progression of excessive weight gain or overweight status. Research, although limited, indicates that early treatment is associated with improved long-term success [29].

## ANTHROPOMETRICS TO ASSESS FAT DISTRIBUTION

### Skinfolds

Subcutaneous fat represents approximately 50% of total body fat, which provides an accessible proxy for assessment of body fatness. The 80th and 95th percentiles of skinfold readings are accepted measures of overweight and obesity in children [6]. Caliper measurement of skinfold thickness requires training and should only be administered by experienced staff. Skinfolds can be measured at a variety of body sites and are used in formulas that predict percent body fat (Appendix A2.3) [6].

### Waist Circumference

An additional method of assessing body composition is the measurement of girth of various body sections. Waist circumference provides an indication of trunk or visceral obesity, which is highly correlated in adults with diabetes and heart disease risk. Similar data are not available for the pediatric population. A metal or fiberglass measuring tape with a metric scale is used to measure

the circumference of the waist and hip. The waist circumference is also a useful indicator for determining reduction in fat weight after treatment (Appendix A2.1.3).

### DIRECT MEASUREMENTS OF BODY COMPOSITION

Direct measurements of body fat content can be found using such tools as hydrodensitometry, bioimpedance, or dual-energy x-ray absorptiometry [35]. In Chapter 4 of this text, Gordon discusses the advantages and disadvantages of these various methods, and detailed protocols may be found in Appendix A2.3.

### LABORATORY TESTING — BASIC PANELS

Secondary assessments may include lipid profiles, total cholesterol, insulin, glucose tolerance, glucose, glycohemoglobin, thyroid function, cortisol, and liver enzymes [47].

If the child is diagnosed with a BMI greater than 95th percentile, additional testing may be warranted. Because pediatric overweight (BMI > 95th percentile for age and sex) is associated with many other disease risk factors, practitioners can further define risk by checking a fasting lipid profile (Appendix A2.2) [41]. Other potentially useful biochemical markers of comorbidities include liver profile, and fasting glucose and insulin. It should be recognized that there are not standardized guidelines for biochemical evaluation, especially because identification of biochemical abnormalities will often not change therapeutic interventions.

### MEASUREMENTS OF ENERGY EXPENDITURE

In some cases, measurement of resting energy expenditure may be useful to help caloric intake goals. Resting energy expenditure can be measured through indirect calorimetry. However, it is difficult to obtain an accurate measure because it is affected by diet, exercise, body temperature, growth, and development [55]. In an outpatient setting, it is difficult to optimally control these factors, and thus measurements can at best be viewed as approximations. Therefore, routine assessment of resting energy expenditure is of limited value.

### REFERRALS

Overweight children with more severe medical problems such as obstructive sleep apnea, obesity hypoventilation syndrome, and orthopedic problems may benefit from more aggressive dietary strategies. If a referral is made to an overweight treatment center, it is important to identify a program that is staffed by medical professionals experienced in the management of these serious comorbidities. Massively overweight children without severe comorbidities, but with a history of weight loss failures, may also benefit from consultation with a pediatric obesity center. Ariza and colleagues [41] provide a detailed assessment and action plan for overweight children in the primary care setting (Figure 7.3).

### GENETICS

Genetic syndromes associated with pediatric obesity include Prader–Willi, Turner syndrome, or Laurence–Moon–Bardet–Biedle [41]. Findings such as developmental delay, short stature/delayed growth, dysmorphic features, abnormal or absent genitalia, and digital anomalies should raise suspicion of an underlying genetic etiology and consideration of definitive testing. If any of these conditions is suspected, referral to a geneticist or other relevant subspecialist is recommended [41].

### ENDOCRINOLOGY

There are several endocrine disorders related to pediatric obesity, including primarily hypothyroidism, type 2 diabetes mellitus, and polycystic ovary disease. Although less common, findings

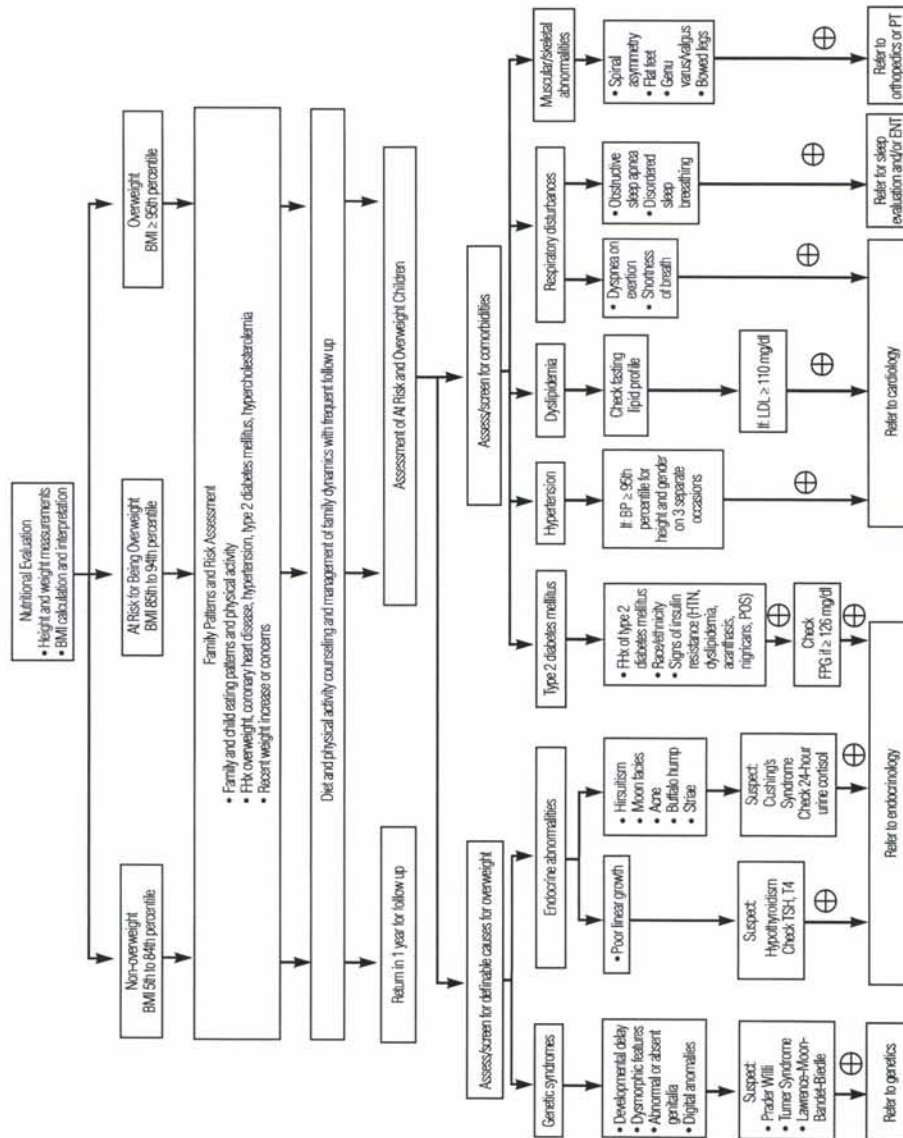


FIGURE 7.3 Primary care assessment and action plan for overweight children. Reprinted with permission from Ariza, A. J., R. S. Greenberg, and R. Unger. Childhood overweight: management approaches in young children. *Pediatr Ann.* 33:33–38, 2004.

indicative of Cushing syndrome, including moon facies, short stature, central obesity, and apparent reduced lean body mass should prompt referral to an endocrinologist. If this is the case, then a 24-hour urine cortisol should be ordered [41].

### Thyroid

Symptoms of hypothyroidism include constipation, cold intolerance, fatigue, and lethargy; signs may include poor linear growth, hypotension, bradycardia, anemia, and loss of deep tendon reflexes. If symptoms or signs of hypothyroidism are present, TSH and T4 levels should be checked, and if these levels are diagnostic of hypothyroidism, the child should be referred to a pediatric endocrinologist [41].

### Pancreas

Insulin resistance is common with excess central (or visceral) adiposity. Insulin resistance is associated with hyperinsulinemia, fatty liver, hypertension, and exercise intolerance. Hyperinsulinemia in children can be associated with normal fasting glucose for some time, but with persistence, there is progression to impaired glucose tolerance, and eventually to  $\beta$ -cell failure and elevated fasting glucose and type 2 diabetes. If insulin resistance or type 2 diabetes mellitus is suspected, fasting insulin and blood glucose levels may be obtained. Hyperglycemia is relatively insensitive until there is frank diabetes, but if fasting glucose levels are 126 mg/dL or more, the child should be referred to a pediatric endocrinologist for further examination [41]. The utility of fasting insulin levels is debated, but documentation of elevation is a finding that motivates some families to make changes, especially if there is a strong family history of type 2 diabetes [41]. An elevated fasting insulin is also part of the constellation of findings referred to as metabolic syndrome [56].

### CARDIOLOGY

During the medical examination, if the child's blood pressure is in the 95th (or higher) percentile for height and gender on three separate occasions, then referral to a cardiologist is suggested [41]. Chest pain is another symptom that may require referral. Treatment of hyperlipidemia (LDL  $\geq$  110 mg/dl) or dyslipidemia (e.g., metabolic syndrome), including initial diet therapy, may also be available through preventive cardiology services, or through nutrition or endocrine subspecialists [41].

### PULMONARY

Pulmonary disorders associated with significant obesity that may require rapid weight loss are obstructive sleep apnea and obesity hypoventilation syndrome [57]. Symptoms indicative of sleep disturbances include snoring, restless sleep, inability to sleep supine, and daytime somnolence. Assessment ideally includes an electrocardiogram to rule out cardiomegaly, sinus dysrhythmias, and right-side heart failure, as well as a sleep study with polysomnography to monitor for hypoxia and cardiac function. Treatment may include supplemental oxygen or positive airway pressure, but at least modest weight loss will also be advantageous. Clinicians should seek guidance from pediatric pulmonologists and obesity treatment specialists [29].

One of the most common pulmonary disorders associated with pediatric obesity is asthma [58]. Asthma is a major cause of chronic pediatric illness and school absenteeism. Moreover, urban minority children with asthma are significantly more overweight than those without asthma [59]. Results of most studies in children do not support a direct causal link between asthma and overweight conditions during childhood [60]. Furthermore, there is insufficient evidence to indicate that asthma precedes overweight conditions in children. Because excess weight exacerbates asthma symptoms, especially during exercise, overweight children with asthma should be monitored closely by a pulmonary specialist.

### ORTHOPEDICS AND PHYSICAL THERAPY

There are several serious orthopedic complications that result from significant obesity during childhood. These include slipped capital femoral epiphysis (manifested as hip or knee pain and limited hip range of motion) and Blount's disease (tibia vara) [29]. Referral should be made to an orthopedic surgeon if radiography confirms either of these conditions [29]. Other related comorbidities include spinal asymmetry, flat feet, genu varus/valgus, Legg–Calve–Perthe disease, and degenerative arthritis. Referral may be made to a physical therapist for an initial evaluation and, in many cases, for therapeutic strategies.

### PSYCHOLOGY

Several psychological disorders are associated with pediatric obesity. Binge-eating disorder should be suspected if the patient reports feeling unable to control food consumption. Depression is commonly found in overweight children, especially in older youth with severe overweight conditions. Jonides and colleagues [61] suggest that the emotional stability of the child and the family will likely determine successful treatment outcomes. If the child displays sadness or reports insomnia, restlessness, or hopelessness, then referral to a psychologist is essential to confirm the diagnosis [29]. In Chapter 9 of this volume, Johnson and von Almen detail appropriate psychological assessment for overweight children.

### SOCIAL SERVICES

The negative effects of food restriction or verbal prompting to consume served food were recently highlighted by the American Academy of Pediatrics [2]. In extreme cases, in which parental behavior results in either food restriction and eating disorders or continued overconsumption and morbid, life-threatening obesity, it may be necessary to refer the patient's family to social services. Likewise, if there is evidence of physical or sexual abuse related to the child's overweight condition, social services should be consulted.

### NUTRITION

Adequate nutrition is vital to growth and development, and both insufficient and imbalanced food consumption can cause nutrient deficiencies, impaired cognitive development, and growth velocity delays [62]. Therefore, if the child's weight condition warrants dietary intervention, referral should be made to a registered dietician. He or she will apply U.S. Department of Agriculture caloric and nutrient guidelines based on the child's age, gender, and medical condition when prescribing a weight-loss plan. A priority for dietary counseling is parent nutrition education so that family-wide changes in food selection and preparation are encouraged [41] (Appendix A3).

### EXERCISE

Pediatric health care professionals should encourage families to engage in regular physical activity to help children achieve and maintain a healthy weight [64]. Local information concerning activity centers, YMCAs, Boys and Girls Clubs, parks, and other recreational areas should be provided to parents [41]. In older children with significant obesity, structured exercise guidelines are useful [64]. Referral to a trained and certified pediatric exercise physiologist will ensure age-appropriate physical activities for the patient.

### EDUCATION

It is now widely accepted that anticipatory guidance on healthy eating habits and physical activity should begin early and for all children (Table 7.2). Readiness for change is essential, and families resistant to lifestyle modification should be referred to a family therapist [34]. Family histories of

**TABLE 7.2**  
**Guidelines for Treatment**

Intervention should begin early, with clinicians initiating treatment when children 3 or fewer years of age become overweight. Because the risk of persistent obesity increases with age, it is crucial for the primary care provider to begin treatment as soon as possible [29].

The physician should ensure that the family is ready for change. It is crucial from the outset that physicians, parents, and children have mutually agreeable goals [34]. If the family seems resistant to change, a referral to a therapist who can address the family's readiness may be needed.

The clinician should educate families about the long-term risks and medical complications of obesity. A family history of obesity related disorders will identify children at particular risk [29].

The clinician should involve the family and all caregivers in the treatment program. Doing so will create new family behaviors consistent with the child's new eating and activity goals, which is important for the long-term success of the treatment [29].

Treatment programs should be focused on making permanent changes in physical activity and eating habits. Gradual, long-term changes have higher success rates than multiple, frequent changes [29].

**TABLE 7.3**  
**Recommended Steps for Monitoring Patients for Overweight and Obesity**

Identify patients who may be at risk by looking at such factors as family history, birth weight, or socioeconomic, ethnic, cultural, or environmental factors.

Calculate and plot body mass index once a year for all children and adolescent patients to identify those who are obese, overweight, or at risk.

Look for changes in body mass index to identify rates of excessive weight gain relative to linear growth.

Monitor patients for risks of obesity-associated chronic diseases such as hypertension, hyperinsulinemia, impaired glucose tolerance, dyslipidemia, and symptoms of obstructive sleep apnea syndrome [2].

obesity and related disorders increase the child's risk of developing comorbid diseases, and the medical consequences of such diseases should be addressed with the family [29]. Treatment success rates improve with participation by family members and care givers. Gradual, permanent changes to the diet and physical activity patterns of the child are more successful than transient, short-term changes (Table 7.2) [29,34].

The BMI measurements of patients should be evaluated each year, with increased attention paid by the physician to patterns of excessive weight gain relative to linear growth as well as to children identified as at greater risk for overweight and obesity (Table 7.3) [2]. Patients at risk of developing obesity-related comorbidities should be monitored closely for signs of these diseases. Pediatricians and health care providers should routinely encourage parents in the healthy dietary practices of breastfeeding, moderation, and appropriate portion sizes, regular fruit and vegetable consumption, limits on sweetened beverages, and other nutritious food choices (Table 7.4) [2]. Pediatricians should also educate parents and caregivers on their roles in establishing physical activity patterns [2]. Increased physical activity and setting limits on sedentary behaviors should also be promoted. The success of prevention efforts will be more favorable if both dietary and physical activity interventions are emphasized (Table 7.4).

## FOLLOW-UP

### AT RISK FOR OBESITY

Children who are diagnosed with one or more primary risk factors or fall above the 85th percentile on the BMI percentiles may benefit from monitoring more frequently than annually (e.g., every

**TABLE 7.4**  
**Preventive Strategies**

Mothers should be encouraged to breastfeed their babies.

Parents and caregivers should be educated in healthy eating patterns as well as on their role in modeling, offering, and regulating food choices.

Families should be educated and given guidance about the effect that they have on their child's development of lifelong physical activity and nutritious eating habits.

Pediatricians should encourage dietary practices that focus on moderation rather than overconsumption, and healthful choices rather than restrictive eating patterns.

Routine physical activity should be consciously promoted by the physician.

Physicians should suggest setting limits for sedentary activities such as watching television or playing video games to less than 2 hours per day.

Steps for optimal prevention should include a combination of dietary and physical activity interventions [2].

6 months) to check for continued upward shifts in BMI [2,36,41]. In addition, anticipatory guidance and brief negotiations for behavior change with parents should be undertaken to change risk eating and physical activity habits.

#### OVERWEIGHT

Families of overweight children require consistent feedback from pediatric health care professionals to determine whether nutrition and physical activity recommendations are providing successful outcomes. Height, weight, and BMI should be calculated regularly (e.g., every 3 months).

#### SEVERE OVERWEIGHT

Severely overweight youth require intense interventions and regular medical monitoring. This is ideally provided by a multidisciplinary team with experience working with pediatric patients and their families. Patients should be examined at least monthly, especially if they are following a calorie-restricted diet. Monitoring to determine the effect of the weight loss plan on lipid profiles and diabetes risk should be conducted quarterly. An expert committee has provided an algorithm to assist pediatric health care professionals with monitoring [29]. In general, a one-half pound/week weight loss is optimal.

#### SUMMARY

For all children, parental education in the medical office setting is strongly recommended regardless of the child's current weight condition, but especially if the parents are obese. Children who are at risk for overweight by virtue of family history or other predisposing factors become increasingly more susceptible as they mature. Thus, age-appropriate, targeted, family-based dietary and physical activity preventive strategies should be consistently promoted, and basic therapeutic interventions should be made available in pediatric clinical settings. The economic burden of obesity-associated illness during childhood in the United States has increased by 43% in the last two decades [66]. Cost-effective individual and group treatment approaches are available and should be both encouraged and financially supported by the pediatric medical community. Academic programs that work simultaneously to conduct research, provide training opportunities for pediatric professionals, and evaluate ongoing interventions to prevent and treat overweight children are also vitally important to reverse this pediatric epidemic.

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## The Impact of Maternal Obesity on Maternal and Fetal Health

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*The increasing rate of maternal obesity provides a major challenge to obstetric practice. Maternal obesity can result in negative outcomes for both women and fetuses. The maternal risks during pregnancy include gestational diabetes and preeclampsia. The fetus is at risk for stillbirth and congenital anomalies. Obesity in pregnancy can also affect health later in life for both mother and child. For women, these risks include heart disease and hypertension. Children have a risk of future obesity and heart disease. Women and their offspring are at increased risk for diabetes. Obstetrician-gynecologists are well positioned to prevent and treat this epidemic.*

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The worldwide prevalence of obesity has increased substantially over the past few decades. Economic, technologic, and lifestyle changes have created an abundance of cheap, high-calorie food coupled with decreased required physical activity. We are eating more and moving less. There is evidence for metabolic dysregulation among obese individuals that has been linked with a number of possible environmental factors, including contaminants from modern industry. Obesity is a significant public health concern and is likely to remain so

for the foreseeable future. Maternal obesity increases the risk of a number of pregnancy complications, including preeclampsia, gestational diabetes mellitus (GDM), and cesarean delivery (Table 1).<sup>1</sup> Excessive weight gain during pregnancy and postpartum retention of pregnancy weight gain are significant risk factors for later obesity in women.<sup>2</sup> Additionally, maternal health can have a significant impact on the in utero environment and, thus, on fetal development and the health of the child later in life (Table 1).<sup>3</sup>

According to the in utero fetal programming hypothesis (Barker hypothesis), size at birth is related to the risk of developing disease later in life.<sup>4</sup> Although the Barker hypothesis originally focused on low birth weight, there is evidence that high birth weight may have its own set of complications later in life. A link between maternal obesity in the first trimester and obesity in children has been demonstrated. Whitaker<sup>5</sup> found that the relative risk of childhood obesity associated with maternal obesity in the first trimester of pregnancy was 2.0 (95% confidence interval [CI], 1.7-2.3) at 2 years of age, 2.3 (95% CI, 2.0-2.6) at 3 years of age, and 2.3 (95% CI, 2.0-2.6) at 4 years of age. Birth weight has also been shown to be directly correlated with body mass index (BMI) later in life.<sup>6</sup>

One mechanism thought to underlie these relationships is in utero fetal programming by nutritional stimuli. Fetuses have to adapt to the supply of nutrients crossing the placenta whether a deficit or an overabundance, and these adaptations may permanently change their physiology and metabolism.<sup>3</sup> These programmed changes may serve as the origins of a diverse array of diseases that arise later in life, including heart disease, hypertension, and non-insulin-dependent diabetes (Figure 1). Moreover, because of fetal programming, obesity may become a self-perpetuating

**Table 1**  
**Obstetric Complications in Obese Pregnant Women**

Complication	OR (95% CI) or % vs Normal Weight	P
Early pregnancy		
Spontaneous abortion (miscarriage)		
After spontaneous conception	1.2 (1.1-1.5)	.04
After IVF conception	1.8 (1.1-3.0)	< .05
Recurrent miscarriage	3.5 (1.1-21.0)	.04
Congenital anomalies		
Neural tube defects	1.8 (1.1-3.0)	< .05
Spina bifida	2.6 (1.5-4.5)	< .05
Congenital heart disease	1.2 (1.1-1.3)	< .05
Omphalocele	3.3 (1.0-10.3)	< .05
Late pregnancy		
Hypertensive disorder of pregnancy		
Gestational nonproteinuric hypertension	2.5 (2.1-3.0)	< .0001
Preeclampsia	3.2 (1.8-5.8)	.007
Gestational diabetes mellitus	2.6 (2.1-3.4)	< .001
Preterm birth	1.5 (1.1-2.1)	< .05
Intrauterine fetal demise (stillbirth)	2.8 (1.9-4.7)	< .001
Peripartum		
Cesarean delivery	47.7% vs 20.7%	< .01
Decreased VBAC success	84.7% vs 66%	.04
Operative morbidity	33.8% vs 20.7%	< .05
Anesthesia complications		
Excessive blood loss		
Postpartum endometritis		
Wound infection/breakdown		
Postpartum thrombophlebitis		
Fetal/neonatal complications		
Fetal macrosomia (EFW ≥ 4500 g)	2.2 (1.6-3.1)	< .001
Shoulder dystocia	3.6 (2.1-6.3)	< .001
Birth weight > 4000 g	1.7 (1.4-2.0)	.0006
Birth weight > 4500 g	2.0 (1.4-3.0)	< .0001
Childhood obesity	2.3 (2.0-2.6)	< .05

95% CI, 95% confidence interval; EFW, estimated fetal weight; IVF, in vitro fertilization; OR, odds ratio; VBAC, vaginal birth after cesarean.

problem. Daughters of obese women may themselves be vulnerable to becoming obese and more likely to have offspring who share this vulnerability.

**Definitions of Obesity**

The most commonly used measurement for defining obesity is BMI, which refers to an individual's weight

in kilograms divided by the square of his or her height in meters. Individuals are deemed *overweight* when they have a BMI between 25 and 30 kg/m<sup>2</sup>; *obesity* is defined as a BMI greater than or equal to 30 kg/m<sup>2</sup>, and *extreme obesity* is defined as a BMI greater than or equal to 40 kg/m<sup>2</sup>. It is important to note, however, that BMI can be misleading. For example,

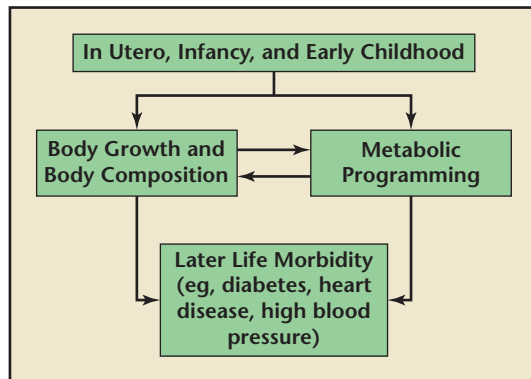


Figure 1. The impact of malnutrition during early development.

weight lifters and professional athletes tend to have high BMI because they have a high muscle mass, not excess fat. These individuals are not at risk for metabolic health problems because the health consequences of obesity come from excess adipose tissue, not the size of one's body. Despite this limitation, BMI continues to be used today because it is easily calculated and is the best tool available from a broad-based health policy perspective.<sup>7</sup>

### Biology of Adipose Tissue

Fat (lipid) is an essential tissue and performs multiple and diverse functions, including providing nutritional, hormonal, and even structural support. The main fat depots in the body are in adipose tissue. Adipocytes are cells specifically adapted for fat storage, serve as a future energy source, and help to avoid the negative metabolic consequences of excess cellular lipid deposits in organs such as mus-

cle, liver, and heart. However, adipose tissue is not a passive organ. It actively regulates metabolism through multiple distinct but overlapping pathways. Adipose tissue also contains a large number of nonfat cells, including fibroblasts and immune cells such as mast cells, macrophages, and leukocytes.<sup>8</sup> Both adipocytes and nonfat cells synthesize and secrete numerous peptide and steroid hormones as well as cytokines and chemokines, and such factors are known to influence local and systemic physiology (Table 2).<sup>8,9</sup> In this way, adipose tissue functions as an endocrine organ,<sup>9</sup> and it is the metabolic function of adipose tissue that causes much of the pathology associated with obesity.

Adipose tissue functions as an endocrine organ in a number of ways. It stores and releases preformed steroid hormones, converts precursors to biologically active hormones, and converts active hormones to inactive metabolites. To this end, adipocytes express a number of enzymes critical

Table 2  
Enzymes and Hormones Produced by Adipose Tissue

Enzyme/Hormone	Function	Changes Associated With Obesity
Aromatase	Converts androgens to estrogens	No change with obesity, but increased fat mass results in greater total conversion
17β-hydroxysteroid hydrogenase	Converts estrone to estradiol and androstendione to testosterone	No change
5α-reductase	Inactivates cortisol	No change
11β-hydroxysteroid dehydrogenase type 1	Converts cortisone to cortisol	Activity is increased in obese women
Leptin	Affects food intake, timing of puberty, bone development, and immune function	Circulating leptin levels are increased in obese women
Tumor necrosis factor α (TNFα)	Represses genes involved in the uptake and storage of nonesterified fatty acids and glucose	Expression of TNFα is increased in the adipose tissue of obese women
Adiponectin	Enhances insulin action	Circulating levels of adiponectin are decreased in obese women

to steroid hormone biosynthesis and metabolism (Table 2). For example, estrone is converted to estradiol in peripheral adipose tissue. Indeed, most if not all circulating estradiol in postmenopausal women comes directly from adipose tissue.<sup>9</sup> Adipose tissue expresses 11 $\beta$ -hydroxysteroid dehydrogenase type 1 (11 $\beta$ -HSD1), which converts cortisone to cortisol, as well as 5 $\alpha$ -reductase, which converts cortisol to 5 $\alpha$ -tetrahydrocortisol. Thus, adipose tissue regulates the local concentration of glucocorticoids and contributes to their metabolic clearance. Finally, adipose tissue secretes a large number of bioactive peptides and cytokines, collectively known as adipokines (Table 2).

Fat in our diet and on our bodies is beneficial as long as it exists in moderation. Too much fat becomes maladaptive, and normal physiology pushed beyond adaptive function becomes pathology, a concept referred to as *allostatic overload*. In the setting of obesity, pathology develops because of an increase in adipose tissue beyond the tolerable functional range. In this way, the metabolic consequences of obesity are analogous to the endocrine dysfunction seen in hyperplasia of any endocrine organ. Consider for a moment the metabolic and health consequences if a person's liver, thyroid, or adrenal gland doubled in size.

### Source of Data on Obesity

The primary source of national data on obesity and overweight in the United States is the National Health and Nutrition Examination Survey (NHANES), which includes both an extensive take-home questionnaire and a physical examination in a mobile examination center (<http://www.cdc.gov/nchs/about/major/nhanes/hlthprofess.htm>). A key feature of NHANES is that it allows for standardized measurements of height and

weight, and, thus, an accurate calculation of BMI. Another source of obesity data is the Pregnancy Risk Assessment Monitoring System (PRAMS), an ongoing population-based surveillance system that examines trends in prepregnancy obesity by maternal demographic and behavioral characteristics. PRAMS collects self-reported data from maternal questionnaires on behaviors associated with pregnancy (<http://www.cdc.gov/prams>). The National Vital Statistics System (NVSS) (<http://www.cdc.gov/nchs/nvss.htm>) contains data on all births in the United States as reported on state birth certificates and is an easy way to collect an abundance of data. Unfortunately, all of these data sources have their limitations. For example, PRAMS only includes 9 states (which represent 18.5%, or 1 in 5, of all live births in the United States<sup>10</sup>) and tracks trends in obesity only over a 10-year period. Similarly, national birth certificate data collected by the NVSS includes maternal weight but not height, and so BMI cannot be calculated.

### Patterns of Maternal Obesity

Data from PRAMS has shown that the prevalence of prepregnancy obesity increased by 69% over a 10-year period, from 13% in 1993-1994 to 22% in 2002-2003.<sup>10</sup> In this report, maternal obesity increased across all categories of age; race; education;

smoking status; Special Supplemental Nutrition Program for Women, Infants, and Children enrollment; and parity.<sup>10</sup>

The Institute of Medicine (IOM) and the National Heart, Lung, and Blood Institute of the National Institutes of Health established guidelines for healthy ranges of weight gain in pregnancy (Table 3). PRAMS data showed that only 1 out of 3 women had weight gain consistent with the recommendations of the IOM.<sup>11</sup> Racial and ethnic factors clearly affect weight gain during pregnancy. According to Brawarsky and colleagues,<sup>12</sup> African American women are more likely to be overweight prior to pregnancy and were most likely to gain weight in excess of the IOM guidelines, white females were most likely to report target weight gain, Hispanic women were least likely to report target gains, and Asian women were more likely to gain less than the recommend weight.

The postpartum period may be a critical time for long-term weight gain and the development of maternal obesity. Excess weight gain during pregnancy and persistent weight retention 1 year postpartum are strong predictors of overweight a decade or more later.<sup>2</sup> According to the National Maternal and Infant Health Survey, more than 30% of women retained 14 lb or more when compared with their recall of their prepregnancy weight, with African American women

**Table 3**  
Recommendations for Weight Gain in Pregnancy

Body Mass Index	Recommended Weight Gain
18.5-24.9 kg/m <sup>2</sup> (normal weight)	25-35 lb (11.2-15.9 kg)
25-29.9 kg/m <sup>2</sup> (overweight)	15-25 lb (6.8-11.2 kg)
> 30 kg/m <sup>2</sup> (obese)	15 lb (6.8 kg)

reporting a larger weight gain during pregnancy and less weight loss postpartum.<sup>13</sup> A more recent study showed that 12% of women retained at least 11 lb 1 year postpartum.<sup>14</sup> These women were more likely to have gained excessive weight during pregnancy and to be younger, heavier prior to pregnancy, nonwhite, unmarried, primiparous, and of lower socioeconomic status. For multiparous women, weight retention from previous pregnancies and the quality of health care received between pregnancies appear to be important deter-

minants of subsequent prepregnancy weight.<sup>14</sup> Some authorities have suggested that more intensive postpartum care in women who are overweight or obese (such as graded exercise and weight loss programs) may be able to significantly impact subsequent pregnancy outcome, but this remains to be definitively demonstrated. Importantly, in a large epidemiologic study in Sweden, an increase in interpregnancy BMI (by at least 3 kg/m<sup>2</sup>) was associated with a higher risk of adverse pregnancy outcomes.<sup>15</sup>

### Effect of Obesity on Maternal Complications in Pregnancy

Maternal obesity increases the risk of a number of pregnancy complications (Table 1) and, as such, requires adjustment to routine prenatal care (summarized in Table 4). Maternal obesity is a risk factor for spontaneous abortion (for both spontaneous conceptions and conceptions achieved through assisted reproductive technology), as well as for unexplained stillbirth (intrauterine fetal demise). A recent meta-analysis of 9 studies revealed that obese pregnant women

**Table 4**  
**Adjustments to Routine Prenatal Care in Obese Pregnant Women**

Risk Factor	Recommended Care
Increased risk of neural tube defect	<ul style="list-style-type: none"> <li>• Preconception folic acid supplementation (4 mg daily ideally 3 months prior to pregnancy through the first trimester)</li> <li>• Maternal serum AFP (15-20 weeks)</li> <li>• Detailed fetal anatomy survey (18-20 weeks)</li> </ul>
Increased risk of hypertensive disorders of pregnancy, including preeclampsia	<ul style="list-style-type: none"> <li>• Baseline 24-hour urinalysis in second trimester</li> <li>• Baseline liver and renal function tests in second trimester</li> <li>• Blood pressure and urine dip for protein at each prenatal visit</li> <li>• There is no effective way to prevent preeclampsia</li> </ul>
Increased risk of gestational diabetes (GDM)	<ul style="list-style-type: none"> <li>• Consider early screening with 1-hour nonfasting 50-g glucose load test (GLT) at 16-20 weeks. If positive, check a definitive 3-hour 100-g glucose tolerance test (GTT) to confirm the diagnosis of GDM. If negative, repeated GLT at the usual gestational age of 24-28 weeks</li> </ul>
Increased risk of unexplained stillbirth	<ul style="list-style-type: none"> <li>• Consider weekly antepartum fetal testing with NST and/or BPP beginning at 36 weeks, especially in women with a BMI <math>\geq</math> 40 kg/m<sup>2</sup> (although this has not been shown to definitively improve perinatal outcome)</li> </ul>
Increased risk of anesthesia complications	<ul style="list-style-type: none"> <li>• ACOG recommends a prelabor or early intrapartum anesthesia consultation for all women with a BMI <math>\geq</math> 40 kg/m<sup>2</sup></li> <li>• Consider early epidural placement in labor</li> <li>• Recheck epidural placement if the patient is transferred to the operative room for cesarean delivery because of increased risk of migration of the epidural catheter</li> </ul>
Failure to lose weight after delivery is associated with subsequent adverse maternal health problems, including complications of future pregnancies	<ul style="list-style-type: none"> <li>• Continue nutrition counseling and exercise program after delivery</li> <li>• Consider consulting a weight loss specialist to optimize postpartum weight loss before attempting another pregnancy</li> <li>• If complicated by GDM, check 2-hour 75-g GTT at or after 6-week postpartum visit</li> </ul>

ACOG, The American College of Obstetricians and Gynecologists; AFP,  $\alpha$ -fetoprotein; BMI, body mass index; BPP, biophysical profile; NST, non-stress test.

have an estimated risk of stillbirth that is twice that of normal weight pregnant women.<sup>16</sup> Several mechanisms have been proposed for this relationship, including the increased risks of hypertensive disorders and gestational diabetes that are associated with maternal obesity during pregnancy.

Maternal obesity is associated with an increased risk of hypertensive disorders of pregnancy, including preeclampsia (gestational proteinuric hypertension), with an odds ratio (OR) of between 2 and 3.<sup>17</sup> The risk increases linearly as BMI increases. For each increase in BMI of 5 to 7 kg/m<sup>2</sup>, there is a corresponding 2-fold increase in the risk of developing preeclampsia.<sup>18</sup>

Obese women are at increased risk of complications at the time of labor and delivery. The rate of successful vaginal delivery decreases progressively as maternal BMI increases. A meta-analysis of 33 studies showed that the ORs of cesarean delivery were

1.46 (95% CI, 1.34-1.60), 2.05 (95% CI, 1.86-2.27), and 2.89 (95% CI, 2.28-3.79) among overweight, obese, and severely obese women, respectively, compared with normal weight pregnant women.<sup>19</sup> According to Ehrenberg and coworkers, the cesarean delivery rate for women weighing less than 200 lb was 18%, versus 39.6% in women who were classified as extremely obese.<sup>20</sup> This 2- to 3-fold increase in cesarean delivery rate is true for both primigravid and multigravid women.<sup>1</sup> Whether this is secondary to increased fetal size or another maternal characteristic is not known.

Maternal obesity also influences the success rate of attempted vaginal birth after cesarean (VBAC). Carroll and colleagues<sup>21</sup> found that women weighing less than 200 lb had a VBAC success rate of 81.8% compared with 57.1% for women weighing 200 to 300 lb and 13.3% for women heavier than 300 lb. A similar relationship

was observed in a subsequent study using BMI rather than absolute maternal weight, with VBAC success rates ranging from 84.7% in women with a BMI lower than 19.8 kg/m<sup>2</sup> to 54.6% in those with a BMI higher than 30 kg/m<sup>2</sup>.<sup>22</sup>

In addition to an increased rate of operative delivery, obese women are also at increased risk of intraoperative complications, including increased infectious morbidity and thromboembolic events (Table 1). There is also an increased risk of anesthetic complications, such as failed intubation at the time of general endotracheal anesthesia.<sup>23</sup> A number of specific recommendations have been proposed to minimize intraoperative complications in obese pregnant women (summarized in Table 5).

The reason obese pregnant women are more likely to end up with a cesarean delivery is not known, but a theory is that obese women are more likely to experience dysfunctional

**Table 5**  
**Recommendations Before, During, and After Surgery in Obese Pregnant Women**

- Consider preoperative **cardiac evaluation**, especially if the patient has diabetes or chronic hypertension. This should include a baseline electrocardiogram and, if abnormal, an echocardiogram and cardiology consultation.
- Give preoperative **broad-spectrum antibiotics** 20-30 minutes before the skin incision to reduce the risk of postpartum endometritis and wound infection.
- Consider using a **large operating table** (especially if the patient is > 300 lb) and having additional personnel in the delivery room.
- Because of the increased risk of intrapartum blood loss, consider having **additional blood products** available in the operating room.
- If indicated, **tape the pannus** out of the surgical field to facilitate visualization and avoid a through-and-through skin incision.
- **Close the subcutaneous layer**. There is extensive evidence that seroma formation and postoperative wound disruption can be decreased in obese women (defined as adipose layer > 2 cm) if the subcutaneous tissues are closed using layers of running sutures.
- **Avoid subcuticular skin closure** to allow serous fluids from the subcutaneous fat to drain out of the incision rather than accumulate in the subcutaneous layer.
- Place **pneumatic compression stockings** on the lower extremities of all obese parturients prior to and during surgery as prophylaxis against deep vein thrombosis (DVT).
- The compression stockings should remain in place until the patient is fully ambulatory. Additional prophylaxis against DVT with prophylactic low-molecular-weight heparin should be considered in women with a body mass index  $\geq$  40 kg/m<sup>2</sup>.
- **Begin early ambulation** to prevent DVT formation.
- Consider **delaying removal of staples or sutures** for a full week to allow the skin to heal completely.

labor. For example, Vahratian and colleagues<sup>24</sup> found that the rate of cervical dilation in nulliparous women in spontaneous labor decreased as maternal BMI increased. In this study, normal weight women (BMI 19.8-26.0 kg/m<sup>2</sup>) took a median duration of 5.43 hours to dilate from 4 to 10 cm, whereas obese women (BMI > 29.0 kg/m<sup>2</sup>) took 6.98 hours. This appears to be true also in women undergoing induction of labor at term. Nuthalapaty and colleagues<sup>25</sup> demonstrated that, although multiparous women progressed faster during induced labor than nulliparous women, in both groups an increase in maternal weight quartile was associated with a decreased rate of cervical dilation and an increase in the duration of labor. Denison and colleagues<sup>26</sup> showed that a higher maternal BMI in the first trimester and a greater increase in BMI throughout pregnancy were associated with a reduced likelihood of spontaneous labor at term, an increased risk of post-term pregnancy, and an increased rate of intrapartum complications.

### Effect of Maternal Obesity on Perinatal Outcome

Maternal obesity is associated with abnormal fetal growth. Women who are heavier are less likely to have a pregnancy complicated by a small-for-gestational age infant or intrauterine growth restriction, but this protective effect appears to dissipate once the maternal BMI reaches the level of obesity (> 30 kg/m<sup>2</sup>). The major concern in obese pregnant women is fetal macrosomia (defined as an estimated fetal weight of greater than or equal to 4500 g), which appears to be increased 2- to 3-fold in obese parturients.<sup>27</sup> Moreover, there appears to be a dose-dependent relationship between maternal obesity and fetal macrosomia. In a recent meta-analysis, the preva-

lence rates of fetal macrosomia were 13.3% and 14.6% for obese and morbidly obese women, respectively, compared with 8.3% for the normal weight control group.<sup>16</sup> In the United States, the mean birth weight between 1985 and 1998 increased from 3423 to 3431 g among whites and from 3217 to 3244 g among blacks.<sup>28</sup> In Canada during the same time period, the mean birth weight increased from 3391 to 3427.<sup>28</sup> In Denmark, the mean birth weight between 1990 and 1999 increased from 3474 g to 3519 g (an increase of 45 g) and macrosomia rates increased from 16.7% to 20%.<sup>29</sup> During a similar time period (1992-2001) in Sweden, there was a 3% increase in the incidence of large-for-gestational-age newborns (defined as birth weight > 2 standard deviations from the mean for a given gestational age).<sup>30</sup> Although a number of factors may explain this global increase in the prevalence of fetal macrosomia, the prevailing data suggest that maternal obesity is the main factor, followed by maternal diabetes status.<sup>27</sup>

Fetal macrosomia in obese women is associated not only with an increase in the absolute size of the fetus, but also in a change in body composition.<sup>31,32</sup> Sewell and coworkers<sup>31</sup> found that the average fat mass of infants born to mothers with a normal BMI (< 25 kg/m<sup>2</sup>) was 334 g, giving a body fat composition of 9.7%. The offspring of women with a BMI > 25 kg/m<sup>2</sup>, on the other hand, had a mean fat mass of 416 g, or a body fat composition of 11.6%. Of note, the majority of this effect appears to be a result of weight gain during pregnancy. Indeed, prepregnancy BMI appears to account for only 6.6% of the observed variation in infantile fat mass and only 7.2% of body fat composition.<sup>33</sup>

Maternal obesity is associated also with an increased risk of neural tube defect (NTD) in the offspring, even

after controlling for ethnicity, maternal age, education, and socioeconomic status.<sup>34-36</sup> Watkins and coworkers<sup>35</sup> concluded that a 1 kg/m<sup>2</sup> increase in BMI is associated with a 7% increased risk of having an infant with NTD. A recent meta-analysis by Rasmussen and colleagues<sup>36</sup> reported that the OR for delivering an infant with NTD was 1.22 (95% CI, 0.99-1.49), 1.70 (95% CI, 1.34-2.15), and 3.11 (95% CI, 1.75-5.46) among overweight, obese, and morbidly obese women, respectively, compared with normal weight women. The mechanism underlying the increased risk of NTD in pregnancies complicated by maternal obesity is unknown. However, a number of theories have been proposed, including a reduction in the amount of folic acid reaching the developing embryo due to insufficient absorption and greater maternal metabolic demands, chronic hypoxia, and increased circulating levels of triglycerides, uric acid, estrogen, and insulin (due, in part, to increased insulin resistance).<sup>34,35</sup>

### Maternal Obesity and Diabetes

Maternal obesity is associated with an increased risk of diabetes, both pregestational diabetes and GDM.<sup>37,38</sup> Compared with normal weight women (BMI < 25 kg/m<sup>2</sup>), a recent meta-analysis of 20 studies demonstrated that the OR of developing GDM was 2.14 (95% CI, 1.82-2.53), 3.56 (95% CI, 3.05-4.21), and 8.56 (95% CI, 5.07-16.04) among overweight (BMI 25-30 kg/m<sup>2</sup>), obese (BMI > 30 kg/m<sup>2</sup>), and severely obese women (BMI > 40 kg/m<sup>2</sup>), respectively.<sup>38</sup> A recent study found that weight gain in the 5 years prior to becoming pregnant, even at a rate of 1.1 to 2.2 kg per year, increases the risk of developing GDM, and that this was especially true for women who were not initially overweight.<sup>37</sup> In addition to prepregnancy BMI, a number of

other demographic factors affect the incidence of GDM. Hedderon and colleagues<sup>37</sup> found that GDM was more likely in women who were older than 35 years of age and who were of Hispanic or Asian ethnicity. In this cohort, GDM was also more common in women with 12 years or less of schooling and with 2 or more previous live births.

The reason obese women are at higher risk of developing GDM has yet to be fully delineated, but is likely related to an increase in insulin resistance. As a result of the continued production of counterregulatory (anti-insulin) hormones by the growing placenta, insulin resistance increases progressively throughout pregnancy. At any single point in pregnancy, however, obese women have higher insulin resistance (lower insulin sensitivity) than women of normal weight, which results in increased availability of lipids for fetal growth and development.<sup>33</sup> Gene microarray profiling of the placentae of obese women with GDM demonstrates increased expression of genes related to lipid metabolism and transport,<sup>39</sup> which likely accounts for the increase in birth weight and fat mass observed in the offspring of such women.

The development of GDM has a number of adverse maternal and fetal implications. For women, these include an increased risk of hyperglycemia, cesarean delivery, and diabetes in later life, with more than 50% of women with GDM acquiring diabetes within 20 years of delivery.<sup>40</sup> The implications for the offspring may be even more severe. Pregnancies complicated by GDM have a 4-fold increased risk of perinatal mortality and a 3-fold increased risk of macrosomia. In addition to being larger, infants born of pregnancies complicated by GDM also have significantly larger skin folds at all areas of measurement (triceps, subscapular,

flank, thigh, abdomen) and, as such, are at increased risk of shoulder dystocia and resultant birth injury.<sup>41</sup> Moreover, offspring born of GDM pregnancies are more likely to develop childhood and adult obesity (OR 1.4 [95% CI, 1.2-1.6] for every 1-kg increment in birth weight) as well as type 2 diabetes mellitus.<sup>42</sup>

### Physician Responsibility

With the known adverse consequences of maternal obesity, it is important that physicians address this issue with their patients. Disconcertingly, Honda<sup>43</sup> found that, over a period of 1 year, only 21.3% and 24.5% of adults who visited their physician received advice about diet and exercise, respectively. On a positive note, a recent survey of 900 obstetrician-gynecologists by The American College of Obstetricians and Gynecologists showed that 80% routinely counsel their pregnant patients about weight control, although only 35% believe that such prenatal counseling will significantly affect the incidence of obesity.<sup>44</sup>

### Conclusions

The incidence of maternal obesity and its attendant comorbid conditions (diabetes, cardiovascular disease) continues to increase at an alarming rate, with major public health implications. Not only does maternal obesity affect the woman, but it also impacts the health of the child, leading to increased childhood obesity and diabetes. Despite improvements in our understanding of this endocrinopathy, there are still many barriers to the clinical care for such women. Obstetrician-gynecologists are in a key position to prevent and treat this epidemic. ■

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**Original Research**



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# Dieting and Disordered Eating Behaviors from Adolescence to Young Adulthood: Findings from a 10-Year Longitudinal Study

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## ABSTRACT

**Background** Disordered eating behaviors are prevalent in adolescence and can have harmful consequences. An important question is whether use of these behaviors in adolescence sets the pattern for continued use into young adulthood.

**Objective** To examine the prevalence and tracking of dieting, unhealthy and extreme weight control behaviors, and binge eating from adolescence to young adulthood.

**Design** Population-based, 10-year longitudinal study (Project EAT-III: Eating Among Teens and Young Adults, 1999-2010).

**Participants/setting** The study population included 2,287 young adults (55% girls, 52% nonwhite). The sample included a younger group (mean age  $12.8 \pm 0.7$  years at baseline and  $23.2 \pm 1.0$  years at follow-up) and an older

group (mean age  $15.9 \pm 0.8$  at baseline and  $26.2 \pm 0.9$  years at follow-up).

**Statistical analyses performed** Longitudinal trends in prevalence of behaviors were tested using generalized estimating equations. Tracking of behaviors were estimated using the relative risk of behaviors at follow-up given presence at baseline.

**Results** In general, the prevalence of dieting and disordered eating was high and remained constant, or increased, from adolescence to young adulthood. Furthermore, behaviors tended to track within individuals and, in general, participants who engaged in dieting and disordered eating behaviors during adolescence were at increased risk for these behaviors 10 years later. Tracking was particularly consistent for the older girls and boys transitioning from middle adolescence to middle young adulthood.

**Conclusions** Study findings indicate that disordered eating behaviors are not just an adolescent problem, but continue to be prevalent among young adults. The tracking of dieting and disordered eating within individuals suggests that early use is likely to set the stage for ongoing use. Findings suggest a need for both early prevention efforts before the onset of harmful behavioral patterns as well as ongoing prevention and treatment interventions to address the high prevalence of disordered eating throughout adolescence and young adulthood.

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**D**ieting, unhealthy weight control practices, and binge eating are often associated with poorer dietary intake (1-3). For example, in a population-based sample of Canadian adolescents, Woodruff and col-



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leagues (2) found that overall dietary quality was lower among youth who were dieting and had weight concerns, compared to those not dieting and not weight concerned. Furthermore, dieting and disordered eating behaviors have been found to predict a number of problematic outcomes, including increased risk for weight gain (4-8), obesity (6-8), and eating disorders (9-12) in adolescents (4,6,7,12) and young adults (5,12,13). For example, in an analysis from an earlier study wave on the population participating in our study (8), we found that adolescents engaging in dieting and those reporting unhealthy weight control behaviors were at two to three times greater risk for being overweight 5 years later, compared to adolescents who did not engage in these behaviors at baseline. Given the harmful consequences associated with disordered eating behaviors, it is important to develop interventions aimed at their prevention.

A frequently asked question regards the best timing for interventions aimed at preventing disordered eating. A high prevalence of disordered eating behaviors during adolescence would support early prevention efforts, with the goal of intervening before the onset of these behaviors. If the prevalence of disordered eating remains stable or increases as young people transition throughout adolescence, then prevention efforts should be continued throughout these life transitions. Although cross-sectional studies have shown a high prevalence of disordered eating during early and middle adolescence (14-16), longitudinal studies examining the course of disordered eating from early adolescence to young adulthood are limited in number and scope, and findings have not been consistent, possibly due to small samples and differences in participant characteristics and study methodologies (13,17-22). Information regarding whether or not disordered eating tracks within individuals is also important in setting program priorities. Most research suggests that disordered eating behaviors during early adolescence are predictive of continued use of these behaviors, as well as progression to a clinical eating disorder during later adolescence or young adulthood (18,19,21-25). However, previous studies have limited generalizability given that most have been conducted within predominantly white samples (13,17,18,20,21) of women (19-23), primarily of high socioeconomic status (21,22).

Our study expands upon the existing literature by examining the prevalence and tracking of dieting and disordered eating behaviors, including unhealthy weight control behaviors, extreme weight control behaviors, and binge eating with loss of control, longitudinally during a 10-year transitional period from adolescence to young adulthood, in a large and diverse population-based sample of young women and men. The first aim of the study was to assess the prevalence of dieting and disordered eating as adolescents move into young adulthood. The second study aim was to determine whether these behaviors track within individuals over time. Specifically, we examined whether adolescents who diet and engage in disordered eating behaviors are at increased risk for these behaviors in young adulthood.

## METHODS

### Study Design and Population

Project EAT-III (Eating and Activity in Teens and Young Adults) is a 10-year longitudinal study aimed at examining eating, activity, and weight-related variables among young people. The sample for our study included 1,030 young men and 1,257 young women. One third of participants (29.9%) were in the younger cohort; at baseline they were in early adolescence (mean age  $12.8 \pm 0.7$  years), and at 10-year follow-up they were in early young adulthood (mean age  $23.2 \pm 1.0$  years). Two thirds of participants (70.1%) were in the older cohort; at baseline they were in middle adolescence (mean age  $15.9 \pm 0.8$  years), and at follow-up they were in middle young adulthood (mean age  $26.2 \pm 0.9$  years).

At baseline (EAT-I: 1998-1999), 4,746 junior and senior high school students at 31 public schools in the Minneapolis/St Paul, MN, metropolitan area completed in-class surveys and anthropometric measures (15,26). At 10-year follow-up (EAT-III: 2008-2009) participants were mailed letters inviting them to complete online or paper surveys. Data were also collected at 5-year follow-up and longitudinal trends in weight control behaviors have previously been described (27). The 5-year follow-up data are not included in this analysis to focus on long-term changes in the prevalence and tracking of behavior patterns and determine whether behaviors begun in adolescence predict similar behaviors in young adulthood. All study protocols were approved by the University of Minnesota's Institutional Review Board Human Subjects Committee. Parental consent and written assent from participants was obtained at baseline. For follow-up surveys, participants reviewed a consent form as part of the online survey or were mailed a consent form with their paper survey. Completion of the Project EAT-III survey implied written consent.

At 10-year follow-up, survey data were collected from 66.4% of those for whom correct contact information was available, representing 48.2% of the original cohort, for a final sample of 2,287 young adults. Attrition was not equal across sociodemographic characteristics. When compared to nonrespondents in Project EAT-III, respondents were more likely to be girls, white, and of higher socioeconomic status (SES). Thus, in all analyses, we weighted our data to allow for the longitudinal sample to be more similar to the original cohort, and more representative of an adolescent/young adult population. Data were weighted using the response propensity method (28) where the inverse of the estimated probability that an individual responded at follow-up was used as the weight. We compared responders at follow-up with nonresponders for the variables being examined in this analysis (ie, dieting, unhealthy and extreme weight control behaviors, and binge eating with loss of control). These comparison analyses were stratified by sex and adjusted for SES, ethnicity/race, and nonresponse weights. In all but one case (responding boys reported lower extreme weight control behaviors than nonresponders) there were no significant differences found for the targeted variables at baseline, indicating that the weighting was generally successful in correcting for any response bias. The final weighted sample was 48.4% white, 18.6% African American, 19.6% Asian, 5.9% Hispanic, 3.3% Native American,

and 4.2% mixed or other race/ethnicity and was well distributed across five levels of SES: low (18.0%), middle-low (19.0%), middle (26.2%), middle-high (23.3%) and high (13.5%).

### Measures

Dieting was assessed with the question, "How often have you gone on a diet during the last year? By 'diet' we mean changing the way you eat so you can lose weight." Responses included "never," "one to four times," "five to 10 times," "more than 10 times," and "I am always dieting." As in previous analyses (8), responses were dichotomized into nondieters (responded never) and dieters (other responses) (test-retest agreement [nondieter vs dieter] 92%).

Unhealthy and extreme weight control behaviors were assessed with the question, "Have you done any of the following things in order to lose weight or keep from gaining weight during the past year?" (yes/no for each method). Responses categorized as unhealthy weight control behaviors included "fasted," "ate very little food," "used a food substitute (powder or a special drink)," "skipped meals," and "smoked more cigarettes." Consistent with prior analyses (27), those reporting the use of one or more unhealthy weight control behaviors were coded as using unhealthy weight control behaviors (test-retest agreement [none vs one or more behaviors] 83%). Extreme weight control behaviors included took diet pills, made myself vomit, used laxatives, and used diuretics. Those reporting the use of one or more of these behaviors were coded as using extreme weight control behaviors (test-retest agreement [none vs one or more behaviors] 97%). Unhealthy and extreme weight control behaviors were also examined individually.

Binge eating with loss of control was assessed with the questions: "In the past year, have you ever eaten so much food in a short period of time that you would be embarrassed if others saw you (binge eating)" (yes/no). "During the times when you ate this way, did you feel you couldn't stop eating or control what or how much you were eating" (yes/no) (29). Those reporting yes to both of these questions were coded as engaging in binge eating with loss of control (test-retest agreement 92% [first question] and 84% [second question]).

Sex, age, race/ethnicity, and SES were based on self-report. Participants were asked about their race/ethnicity (test-retest  $\kappa=0.70-0.83$ ) as prior research has identified racial/ethnic differences in weight-related variables among young people (5,30). The prime determinant of SES was the higher education level of either parent (test-retest  $\kappa=0.78$ ) during the administration of the Project EAT-I survey (31).

### Statistical Analysis

Longitudinal trends in the prevalence of different behaviors were estimated and tested using generalized estimating equations (32) to account for correlation within individuals across time. Specifically, for each outcome, log binomial models (33) were used including a main effect for year (1999 or 2010), cohort (younger or

older), and a year by cohort interaction along with control variables including baseline ethnicity/race and SES and nonresponse weights. The time trends in each cohort were tested based on these models by forming contrasts combining the generalized estimating equation estimates of the main effect for year and the year by cohort interaction. Regression-adjusted prevalences of behaviors over time and by cohort were obtained based on the fitted log binomial models using the predicted probability for a person with average values on the control variables.

Tracking of behaviors within-person over time was estimated and tested using log binomial models of follow-up behaviors on baseline behaviors stratified by cohort and sex and controlling for nonresponse weights. Relative risks from these fitted models were used to test tracking and represent how many times more likely an individual was to be doing a behavior at follow-up given that they were doing the behavior at baseline. *P* values and 95% confidence intervals for relative risks were calculated based on the likelihood ratio test. SAS version 9.2 (2010, SAS Institute Inc, Cary, NC) was used for all analyses.

## RESULTS

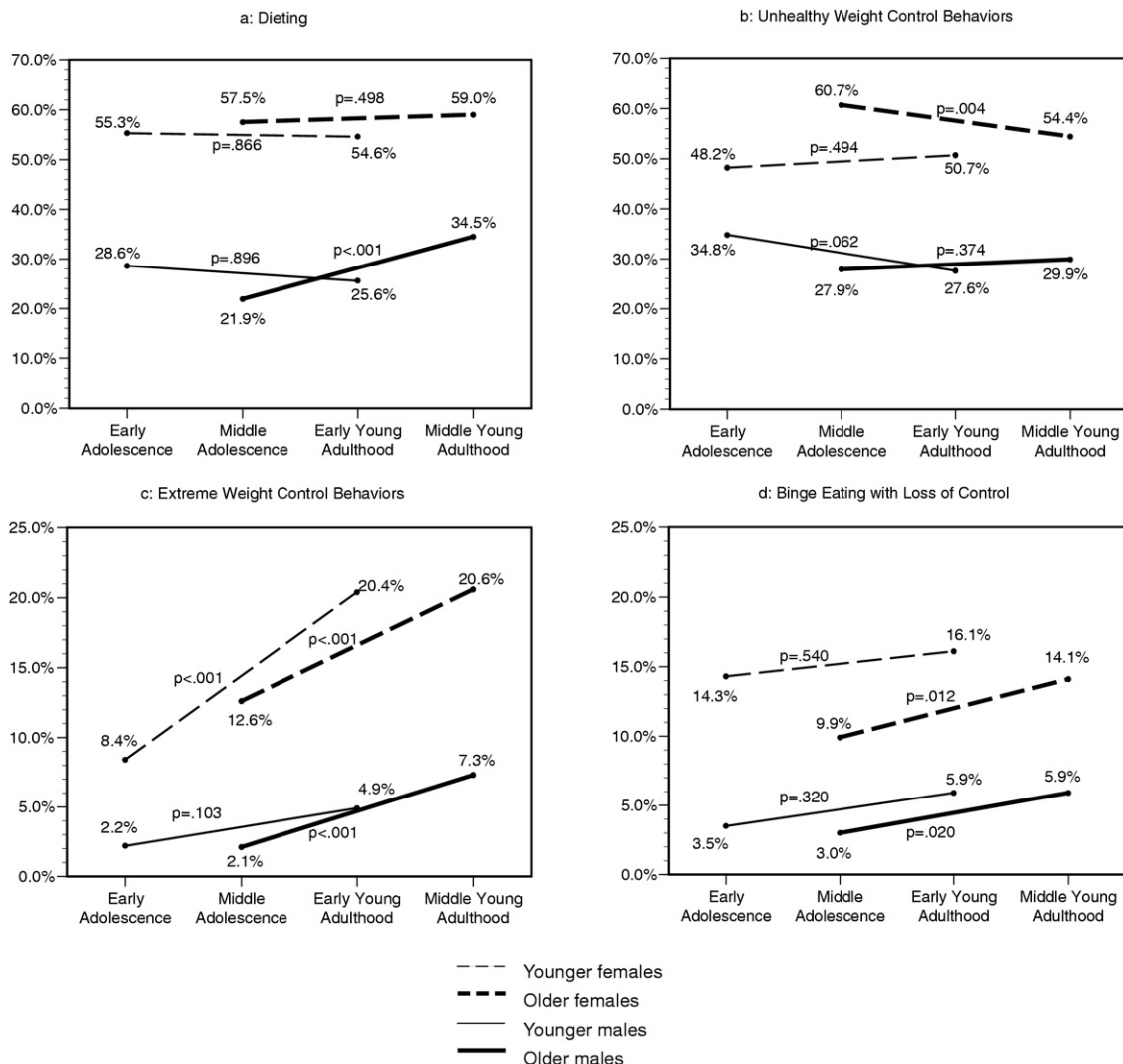
### Prevalence of Dieting and Disordered Eating: Adolescence to Young Adulthood

About half of girls reported dieting in the past year as compared to about a fourth of the boys. Among both age cohorts of girls, the prevalence of dieting remained fairly constant from adolescence through young adulthood (Figure 1). Among boys, the prevalence of dieting stayed constant over time in the younger age cohort, but significantly increased in the older cohort as participants progressed from middle adolescence to middle young adulthood (21.9% to 27.9%,  $P<0.001$ ).

In younger girls, the prevalence of unhealthy weight control behaviors remained constant from early adolescence to early young adulthood (Figure 1). Among older girls, the prevalence of unhealthy weight control behaviors showed a statistically significant decrease from middle adolescence to middle young adulthood, but still remained very high (60.7% to 54.4%,  $P=0.004$ ). Approximately one third of boys reported unhealthy weight control behaviors, and the prevalence remained fairly constant during the study period in both age cohorts.

For extreme weight control behaviors, significant increases from adolescence to young adulthood were found in girls for both age cohorts and for the older cohort of boys (Figure 1). Among girls, the use of extreme weight control behaviors increased from 8.4% to 20.4% ( $P\leq 0.001$ ) between early adolescence and early young adulthood and from 12.6% to 20.6% ( $P<0.001$ ) between middle adolescence and middle young adulthood. For the older boys, extreme weight control behaviors increased from 2.1% in middle adolescence to 7.3% in middle young adulthood ( $P<0.001$ ).

Binge eating increased in the older cohorts of both girls and boys (Figure 1). Among girls, the prevalence increased from 9.9% during middle adolescence to 14.1% in middle young adulthood ( $P=0.012$ ). Among



**Figure 1.** Prevalences of dieting, unhealthy and extreme weight control behaviors, and binge eating with loss of control from adolescence to young adulthood, by age cohort and sex, adjusted for socioeconomic status and ethnicity/race. *P* values test change over time. NOTE: Information from this figure is available online at [www.adajournal.org](http://www.adajournal.org) as part of a PowerPoint presentation.

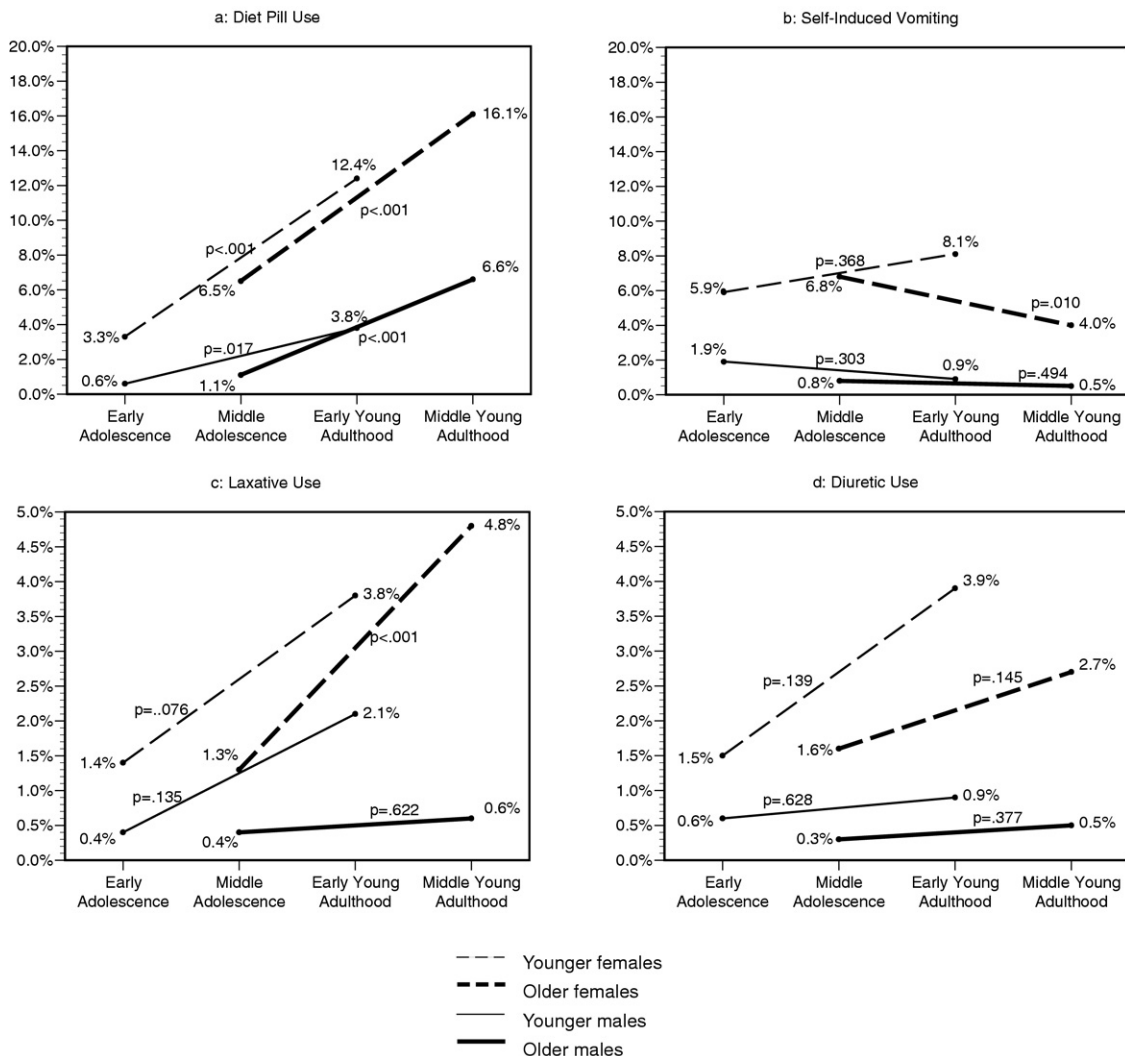
boys, the prevalence of binge eating increased from 3.0% in middle adolescence to 5.9% in middle young adulthood ( $P=0.020$ ). Increases in binge eating among the younger cohorts of boys and girls were not statistically significant.

An examination of the specific types of extreme weight control behaviors revealed significant increases in diet pill use for all age and sex groups over the 10-year study period (see Figure 2). Among girls, diet pill use increased from 3.3% to 12.4% ( $P<0.001$ ) between early adolescence and early young adulthood and from 6.5% to 16.1% ( $P<0.001$ ) between middle adolescence and middle young adulthood. For boys, diet pill use increased from 0.6% to 3.8% ( $P=0.017$ ) between early adolescence and early young adulthood and from 1.1% to 6.6% ( $P<0.001$ ) between middle adolescence and middle young adulthood. Laxative use increased from 1.3% among girls in middle adolescence to 4.8% among girls in middle young adult-

hood ( $P<0.001$ ). In contrast, vomiting decreased from 6.8% during middle adolescence to 4.0% in middle young adulthood ( $P=0.010$ ) among the older cohort of girls. Changes in diuretics were not statistically significant.

### Tracking of Dieting and Disordered Eating from Adolescence to Young Adulthood

Dieting and disordered eating tended to track from adolescence to young adulthood, particularly among the older cohorts of girls and boys transitioning from middle adolescence to middle young adulthood (see the Table). Girls and boys from both age cohorts who dieted in adolescence were significantly more likely to diet in young adulthood, compared to those who didn't diet during adolescence. Similarly, adolescents from all four age and sex groups who used unhealthy weight control behaviors in adolescence were at significantly greater risk for these behav-



**Figure 2.** Prevalences of specific extreme weight control behaviors from adolescence to young adulthood, by age cohort and sex, adjusted for socioeconomic status and ethnicity/race. *P* values test change over time. NOTE: Information from this figure is available online at [www.adajournal.org](http://www.adajournal.org) as part of a PowerPoint presentation.

iors in young adulthood. Use of extreme weight control behaviors during middle adolescence predicted greater risk for these behaviors 10 years later during middle young adulthood for the older cohorts of girls and boys. Finally, binge eating during adolescence increased risk for binge eating during young adulthood for the older cohorts of girls and boys and for the younger girls.

## DISCUSSION

This study found that, in general, the prevalence of dieting and disordered eating behaviors was high and either remained constant or increased from adolescence to young adulthood. Of particular concern was the large increase in extreme weight control behaviors among youth transitioning from adolescence to young adulthood. Diet pill use more than tripled in most of the age and sex groups during the 10-year study period. Of concern, one fifth of female young adults reported the use of extreme

weight control behaviors. Furthermore, behaviors tended to track within individuals and, in general, participants who engaged in dieting and disordered eating behaviors during adolescence were at increased risk for these behaviors 10 years later. Tracking was particularly consistent for the older girls and boys transitioning from middle adolescence to middle young adulthood. The tracking of these potentially harmful behaviors suggests that their use is not just a phase that adolescents go through, but instead indicate that early use of dieting and disordered eating behaviors may set the stage for continued use of these behaviors later on. Together the findings suggest a need for both early prevention efforts before the onset of behavioral patterns that tend to continue over time, as well as ongoing prevention and treatment interventions to address the high prevalence throughout adolescence and young adulthood.

Findings from this study are generally in agreement

**Table.** Tracking of dieting, unhealthy and extreme weight control behaviors, and binge eating during a 10-year period from adolescence to young adulthood by age and sex<sup>a</sup>

Behavior	Younger Females (n=308)			Older Females (n=722)			Younger Males (n=377)			Older Males (n=880)		
	RR	95% CI	P value	RR	95% CI	P value	RR	95% CI	P value	RR	95% CI	P value
Dieting	1.32	(1.09, 1.60)	0.005	1.61	(1.41, 1.83)	<0.001	1.46	(1.03, 2.05)	0.033	2.27	(1.89, 2.72)	<0.001
Unhealthy weight control behaviors	1.54	(1.27, 1.87)	<0.001	1.73	(1.49, 2.01)	<0.001	1.63	(1.19, 2.24)	0.002	2.10	(1.72, 2.56)	<0.001
Extreme weight control behaviors	1.22	(0.68, 2.20)	0.509	2.13	(1.62, 2.80)	<0.001	1.07	(0.11, 10.02)	0.955	6.43	(3.63, 11.37)	<0.001
Binge eating with loss of control	2.21	(1.31, 3.71)	0.003	2.42	(1.68, 3.47)	<0.001	0.47	(0.03, 7.12)	0.586	5.27	(2.68, 10.34)	<0.001

<sup>a</sup>Relative risks (RRs) and 95% confidence intervals (CIs) for engaging in behaviors in young adulthood if engaged in behavior during adolescence as compared to not having engaged in behavior in adolescence.  
NOTE: Information from this table is available online at [www.adajournal.org](http://www.adajournal.org) as part of a PowerPoint presentation.

with the available research that has examined population-based samples of boys and girls from adolescence through young adulthood, although there are some notable differences. Results from this study showing sex differences in the course of disordered eating behaviors are in agreement with a prior 10-year longitudinal study conducted by Heatherton and colleagues among young people who were college students in 1982 (13). Both studies showed dieting and disordered eating behaviors continue to be more prevalent among girls than boys as they transition through adolescence and young adulthood. In addition, both studies found evidence of increases in dieting among boys as they age through their 20s. However, Heatherton and colleagues (13) found that dieting and disordered eating behaviors decreased among females transitioning to middle young adulthood, whereas in our study, the prevalence of these behaviors either increased or remained constant over time. These differences might reflect the use of different measures, secular trends, or real differences between the two sample populations. Heatherton and colleagues (13) conducted their baseline analysis on college students in 1982, nearly 30 years ago, thus secular changes are certainly possible. Furthermore, the sample studied by Heatherton and colleagues (13) was drawn from a selective northeastern college in the United States, and had much higher levels of dieting at baseline (75%) than the Project EAT population (50%). With regard to the tracking of behaviors, Kotler and colleagues (18) examined longitudinal relationships between childhood, adolescent, and adulthood eating disorders over a 17-year interval. Their study population differed from the Project EAT population in that participants were drawn from a rural area and were primarily white. In addition, outcome measures differed across the studies. Nevertheless, findings from both studies are similar in showing that disordered eating behaviors tend to track between adolescence and adulthood.

Study strengths include the population-based nature of the sample; its large size; and its diversity in terms of sex, ethnicity/race, and SES. Much of the previous longitudinal work examining the course of disordered eating or eating disorders have included samples that were either female (19-23), primarily white (13,17,18,20,21), or from higher SES backgrounds (21,22), limiting the generalizability of the findings. The long follow-up period, which captured major periods of transition, is another study strength as few other studies have followed adolescents into middle young adulthood. Finally, the use of two age cohorts allowed for the capturing of slightly different stages of transition and for the replication of findings in different individuals. However, study limitations also need to be taken into account in interpreting the findings. Dieting and disordered eating behaviors were assessed with brief self-reported measures and frequency of use of behaviors was not assessed. The use of diet pills once during the past year, while of concern, is less of a concern than the weekly use of diet pills. In addition, there was attrition from the original study population. For this reason, analyses only included individuals who were present at both EAT-I and EAT-III assessments, and the population was weighted by nonresponse propensity so that the statistical results can be considered more representative of the original school-based sample.

## CONCLUSIONS

The ineffectiveness of dieting for weight gain prevention during adolescence (4-8), the harmful consequences associated with disordered eating behaviors (19,34-37), and the high prevalence of these behaviors during adolescence (21,38-40) have been shown in previous studies. This study adds to this concerning body of literature by demonstrating that the high prevalence of these behaviors continues from adolescence through young adulthood. Furthermore, individuals who begin these behaviors during adolescence are placing themselves at increased risk for their continued use 10 years later. Further research should explore tracking of dieting and disordered eating from adolescence to later stages of adulthood. Research with population-based samples is also needed to identify factors that predict continued use of these behaviors and progression to more serious outcomes such as eating disorders. It is also important to examine the effects of persistent use of dieting and disordered eating behaviors on behavioral, physical, and psychological outcomes such as binge eating, weight status, and depression. Finally, it is crucial to implement and evaluate interventions. Findings from this study, in conjunction with previous studies, argue for early and ongoing efforts aimed at the prevention, early identification, and treatment of disordered eating behaviors in young people. Within clinical practices, dietetics practitioners and other health care providers should be asking about the use of these behaviors before adolescence, throughout adolescence, and into young adulthood. Given the growing concern about obesity, it is important to let young people know that dieting and disordered eating behaviors can be counterproductive to weight management (4-8). Young people concerned about their weight should be provided with support for healthful eating and physical activity behaviors that can be implemented on a long-term basis, and should be steered away from the use of unhealthy weight control practices.

## STATEMENT OF POTENTIAL CONFLICT OF INTEREST:

No potential conflict of interest was reported by the authors.

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THE FOURTH REPORT ON THE

# Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents



U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
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## THE FOURTH REPORT ON THE DIAGNOSIS, EVALUATION, AND TREATMENT OF HIGH BLOOD PRESSURE IN CHILDREN AND ADOLESCENTS

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### FINANCIAL DISCLOSURES

Dr. Flynn has served as a consultant/ advisor for Pfizer Inc., AstraZeneca LP, ESP-Pharma, and Novartis Pharmaceuticals; he received funding/grant support for research projects from Pfizer, AstraZeneca, and Novartis.

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# Contents

<b>FOREWORD</b> .....	<b>VII</b>
<b>INTRODUCTION</b> .....	<b>1</b>
<b>METHODS</b> .....	<b>3</b>
<b>DEFINITION OF HYPERTENSION</b> .....	<b>4</b>
<b>MEASUREMENT OF BLOOD PRESSURE IN CHILDREN</b> .....	<b>5</b>
Ambulatory Blood Pressure Monitoring .....	7
<b>BLOOD PRESSURE TABLES</b> .....	<b>8</b>
Using the Blood Pressure Tables .....	9
<b>PRIMARY HYPERTENSION AND EVALUATION FOR COMORBIDITIES</b> .....	<b>16</b>
<b>EVALUATION FOR SECONDARY HYPERTENSION</b> .....	<b>18</b>
Physical Examination .....	18
Additional Diagnostic Studies for Hypertension .....	19
Renin Profiling .....	19
Evaluation for Possible Renovascular Hypertension .....	19
Invasive Studies .....	19
<b>TARGET-ORGAN ABNORMALITIES IN CHILDHOOD HYPERTENSION</b> .....	<b>22</b>
Clinical Recommendation .....	23
<b>THERAPEUTIC LIFESTYLE CHANGES</b> .....	<b>24</b>
<b>PHARMACOLOGIC THERAPY OF CHILDHOOD HYPERTENSION</b> .....	<b>26</b>
<b>APPENDIX A. DEMOGRAPHIC DATA</b> .....	<b>34</b>
<b>APPENDIX B. COMPUTATION OF BLOOD PRESSURE PERCENTILES FOR ARBITRARY SEX, AGE, AND HEIGHT</b> .....	<b>36</b>
<b>SCHEME USED FOR CLASSIFICATION OF THE EVIDENCE</b> .....	<b>39</b>
<b>REFERENCES</b> .....	<b>40</b>

# List of Tables

<b>TABLE 1.</b>	Conditions Under Which Children <3 Years Old Should Have Blood Pressure Measured	5
<b>TABLE 2.</b>	Recommended Dimensions for Blood Pressure Cuff Bladders	6
<b>TABLE 3.</b>	Blood Pressure Levels for Boys by Age and Height Percentile	10
<b>TABLE 4.</b>	Blood Pressure Levels for Girls by Age and Height Percentile	12
<b>TABLE 5.</b>	Classification of Hypertension in Children and Adolescents, With Measurement Frequency and Therapy Recommendations	14
<b>TABLE 6.</b>	Indications for Antihypertensive Drug Therapy in Children	14
<b>TABLE 7.</b>	Clinical Evaluation of Confirmed Hypertension	15
<b>TABLE 8.</b>	Examples of Physical Examination Findings Suggestive of Definable Hypertension	20
<b>TABLE 9.</b>	Antihypertensive Drugs for Outpatient Management of Hypertension in Children 1–17 Years Old	28
<b>TABLE 10.</b>	Antihypertensive Drugs for Management of Severe Hypertension in Children 1–17 Years Old	33
<b>TABLE A-1.</b>	Demographic Data on Height/Blood Pressure Distribution Curves by Study Population	35
<b>TABLE B-1.</b>	Regression Coefficients From Blood Pressure Regression Models	38

# List of Figures

<b>FIGURE 1.</b>	Management Algorithm	32
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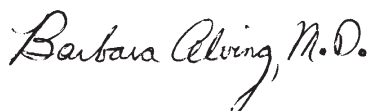
# Foreword

This is the fourth report from the National High Blood Pressure Education Program (NHBPEP) Working Group on Children and Adolescents; it updates the previous publication, *Update on the Task Force Report (1987) on High Blood Pressure in Children and Adolescents (Pediatrics. 1996;98:649–58)*. The purpose of this report is to update clinicians on the latest recommendations concerning the diagnosis, evaluation, and treatment of hypertension in children; recommendations are based on English-language, peer-reviewed, scientific evidence (from 1997 to 2004) and the consensus expert opinion of the NHBPEP Working Group.

This report includes new data from the 1999–2000 National Health and Nutrition Examination Survey (NHANES), as well as revised blood pressure (BP) tables that include the 50th, 90th, 95th, and 99th percentiles by sex, age, and height. Hypertension in children and adolescents continues to be defined as systolic BP (SBP) and/or diastolic BP (DBP) that is, on repeated measurement, at or above the 95th percentile for sex, age, and height. BP between the 90th and 95th percentile in childhood is now termed “prehypertension” and is an indication for lifestyle modifications. New guidelines are provided for the staging of hypertension in children and adolescents, as well as updated recommendations for diagnostic evaluation of hypertensive children. In addition, the report evaluates the evidence of early target-organ damage in children and

adolescents with hypertension; provides the rationale for early identification and treatment; and provides revised recommendations, based on recent studies, for the use of antihypertensive drug therapy. Treatment recommendations also include updated evaluation of nonpharmacologic therapies to reduce additional cardiovascular risk factors. The report describes how to identify hypertensive children who need additional evaluation for sleep disorders that may be associated with BP elevation.

Dr. Bonita Falkner has our deep appreciation for leading the members of the NHBPEP Working Group in developing this new report. Dr. Falkner and the Working Group performed diligently and brilliantly to assemble this document in a timely manner. Applying these recommendations to clinical practice will address the important public health issue of improving inadequate BP control.



Barbara M. Alving, M.D.  
Acting Director  
National Heart, Lung, and Blood Institute  
and  
Chair  
National High Blood Pressure Education Program  
Coordinating Committee



# Introduction

Considerable advances have been made in detection, evaluation, and management of high blood pressure, or hypertension, in children and adolescents. Because of the development of a large national database on normative blood pressure (BP) levels throughout childhood, the ability to identify children who have abnormally elevated BP has improved. On the basis of developing evidence, it is now apparent that primary hypertension is detectable in the young and occurs commonly. The long-term health risks for hypertensive children and adolescents can be substantial; therefore, it is important that clinical measures be taken to reduce these risks and optimize health outcomes.

The purpose of this report is to update clinicians on the latest scientific evidence regarding BP in children and to provide recommendations for diagnosis, evaluation, and treatment of hypertension based on available evidence and consensus expert opinion of the Working Group when evidence was lacking. This publication is the fourth report from the National High Blood Pressure Education Program (NHBPEP) Working Group on Children and Adolescents and updates the previous 1996 publication, *Update on the Task Force Report (1987) on High Blood Pressure in Children and Adolescents*.<sup>1</sup>

This report includes the following information:

- New data, from the 1999–2000 National Health and Nutrition Examination Survey (NHANES), have been added to the child-

hood BP database, and the BP data have been reexamined. The revised BP tables now include the 50th, 90th, 95th, and 99th percentiles by sex, age, and height.

- Hypertension in children and adolescents continues to be defined as systolic BP (SBP) and/or diastolic BP (DBP) that is, on repeated measurement, at or above the 95th percentile. BP between the 90th and 95th percentile in childhood had been designated “high normal.” To be consistent with the *Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure* (JNC 7), this level of BP will now be termed “prehypertensive” and is an indication for lifestyle modifications.<sup>2</sup>
- The evidence of early target-organ damage in children and adolescents with hypertension is evaluated, and the rationale for early identification and treatment is provided.
- Based on recent studies, revised recommendations for use of antihypertensive drug therapy are provided.
- Treatment recommendations include updated evaluation of nonpharmacologic therapies to reduce additional cardiovascular risk factors.
- Information is included on the identification of hypertensive children who need additional evaluation for sleep disorders.



# Methods

In response to the request of the NHBPEP Chair and Director of the National Heart, Lung, and Blood Institute (NHLBI) regarding the need to update the JNC 7 report,<sup>2</sup> some NHBPEP Coordinating Committee members suggested that the NHBPEP Working Group Report on Hypertension in Children and Adolescents should be revisited. Thereafter, the NHLBI Director directed the NHLBI staff to examine issues that might warrant a new report on children. Several prominent clinicians and scholars were asked to develop background manuscripts on selected issues related to hypertension in children and adolescents. Their manuscripts synthesized the available scientific evidence. During the spring and summer of 2002, NHLBI staff and the chair of the 1996 NHBPEP Working Group report on hypertension in children and adolescents reviewed the scientific issues addressed in the background manuscripts as well as contemporary policy issues. Subsequently, the staff noted that a critical mass of new information had been identified, thus warranting the appointment of a panel to update the earlier NHBPEP Working Group Report. The NHLBI Director appointed the authors of the background papers and other national experts to serve on the new panel. The chair and NHLBI staff developed a report outline and timeline to complete the work in 5 months.

The background papers served as focal points for review of the scientific evidence at the first meeting. The members of the Working Group were assembled into teams, and each team prepared specific sections of the report. In developing the focus of each section, the Working Group was asked to consider the peer-reviewed scientific literature published in English since 1997. The scientific evidence was classified by the system used in the JNC 7.<sup>2</sup> The chair assembled the sections submitted by each team into the first draft of the report. The draft report was distributed to the Working Group for review and comment. These comments were assembled and used to create the second draft. A subsequent onsite meeting of the Working Group was conducted to discuss further revisions and the development of the third draft document. Amended sections were reviewed, critiqued, and incorporated into the third draft. After editing by the chair for internal consistency, the fourth draft was created. The Working Group reviewed this draft, and conference calls were conducted to resolve any remaining issues that were identified. When the Working Group approved the final document, it was distributed to the Coordinating Committee for review.

# Definition of Hypertension

- *Hypertension is defined as average SBP and/or DBP that is greater than or equal to the 95th percentile for sex, age, and height on three or more occasions.*
- *Prehypertension in children is defined as average SBP or DBP levels that are greater than or equal to the 90th percentile, but less than the 95th percentile.*
- *As with adults, adolescents with BP levels greater than or equal to 120/80 mmHg should be considered prehypertensive.*
- *A patient with BP levels above the 95th percentile in a physician's office or clinic, who is normotensive outside a clinical setting, has white-coat hypertension. Ambulatory BP monitoring (ABPM) is usually required to make this diagnosis.*

The definition of hypertension in children and adolescents is based on the normative distribution of BP in healthy children. Normal BP is defined as SBP and DBP that is less than the 90th percentile for sex, age, and height. Hypertension is defined as average SBP or DBP that is greater than or equal to the 95th percentile for sex, age, and height on at least three separate occasions. Average SBP or DBP levels that are greater than or equal to the 90th percentile, but less than the 95th percentile, had been designated as “high normal” and were considered to be an indication of heightened risk for developing hypertension. This designation is consistent with the description of “prehypertension” in adults. The JNC 7 Committee now defines prehyper-

tension as a BP level that is equal to or greater than 120/80 mmHg and recommends the application of preventive health-related behaviors, or therapeutic lifestyle changes, for individuals having SBP levels that exceed 120 mmHg.<sup>2</sup> It is now recommended that, as with adults, children and adolescents with BP levels at 120/80 mmHg or above, but less than the 95th percentile, should be considered prehypertensive.

The term white-coat hypertension defines a clinical condition in which the patient has BP levels that are above the 95th percentile when measured in a physician's office or clinic, whereas the patient's average BP is below the 90th percentile outside of a clinical setting.

# Measurement of Blood Pressure in Children

- *Children >3 years old who are seen in a medical setting should have their BP measured.*
- *The preferred method of BP measurement is auscultation.*
- *Correct measurement requires a cuff that is appropriate to the size of the child's upper arm.*
- *Elevated BP must be confirmed on repeated visits before characterizing a child as having hypertension.*
- *Measures obtained by oscillometric devices that exceed the 90th percentile should be repeated by auscultation.*

Children over the age of 3 years who are seen in medical care settings should have their BP measured at least once during every health care episode. Children under age 3 should have their BP measured in special circumstances. (See table 1.)

The BP tables are based on auscultatory measurements; therefore, the preferred method of measurement is auscultation. As discussed below, oscillometric devices are convenient and minimize observer error, but they do not provide measures that are identical to auscultation. To confirm hypertension, the BP in children should be measured with a standard clinical sphygmomanometer, using a stethoscope placed over the brachial artery pulse, proximal and medial to the cubital fossa, and below the bottom edge of the cuff (i.e., about 2 cm above the cubital fossa). The use of the bell of the stethoscope may allow softer Korotkoff sounds to be heard better.<sup>3,4</sup> The use of an appropriately sized cuff may preclude the placement of the stethoscope in this precise location, but there is little evidence that significant inaccuracy is introduced, either if the head of the stethoscope is slightly out of position or if there is contact between the cuff and the stethoscope. Preparation of the child for standard measurement can affect the BP level just as much as

technique.<sup>5</sup> Ideally, the child whose BP is to be measured should have avoided stimulant drugs or foods, have been sitting quietly for 5 minutes, and seated with his or her back supported, feet on the floor and right arm supported, cubital fossa at heart level.<sup>6,7</sup> The right arm is preferred in repeated measures of BP for consistency and comparison to standard tables and because of the possibility of

**TABLE 1**

## **Conditions Under Which Children <3 Years Old Should Have Blood Pressure Measured**

- History of prematurity, very low birthweight, or other neonatal complication requiring intensive care
- Congenital heart disease (repaired or nonrepaired)
- Recurrent urinary tract infections, hematuria, or proteinuria
- Known renal disease or urologic malformations
- Family history of congenital renal disease
- Solid organ transplant
- Malignancy or bone marrow transplant
- Treatment with drugs known to raise BP
- Other systemic illnesses associated with hypertension (neurofibromatosis, tuberous sclerosis, etc.)
- Evidence of elevated intracranial pressure

TABLE 2

Recommended Dimensions for Blood Pressure Cuff Bladders

Age Range	Width (cm)	Length (cm)	Maximum Arm Circumference (cm)*
Newborn	4	8	10
Infant	6	12	15
Child	9	18	22
Small adult	10	24	26
Adult	13	30	34
Large adult	16	38	44
Thigh	20	42	52

\* Calculated so that the largest arm would still allow bladder to encircle arm by at least 80 percent.

coarctation of the aorta, which might lead to false (low) readings in the left arm.<sup>8</sup>

Correct measurement of BP in children requires use of a cuff that is appropriate to the size of the child's upper right arm. The equipment necessary to measure BP in children, ages 3 through adolescence, includes child cuffs of different sizes and must also include a standard adult cuff, a large adult cuff, and a thigh cuff. The latter two cuffs may be needed for use in adolescents.

By convention, an appropriate cuff size is a cuff with an inflatable bladder width that is at least 40 percent of the arm circumference at a point midway between the olecranon and the acromion. (See [www.americanheart.org/presenter.jhtml?identifier=576](http://www.americanheart.org/presenter.jhtml?identifier=576).)<sup>9,10</sup> For such a cuff to be optimal for an arm, the cuff bladder length should cover 80–100 percent of the circumference of the arm.<sup>1,11</sup> Such a requirement demands that the bladder width-to-length ratio be at least 1:2. Not all commercially available cuffs are manufactured with this ratio. Additionally, cuffs labeled for certain age populations (e.g., infant cuffs, child cuffs) are constructed with widely disparate dimensions. Accordingly, the Working Group recommends that standard cuff dimensions for children be adopted. (See table 2.) BP measurements are overestimated to a greater degree with a cuff that is too small than they are underestimated by a cuff that is too large. *If a cuff is too small, the*

*next largest cuff should be used, even if it appears large. If the appropriate cuffs are used, the cuff size effect is obviated.*<sup>12</sup>

SBP is determined by the onset of the “tapping” Korotkoff sounds (K1). Population data in children<sup>1</sup> and risk-associated epidemiological data in adults<sup>13</sup> have established the fifth Korotkoff sound (K5), or the disappearance of Korotkoff sounds, as the definition of DBP. In some children, Korotkoff sounds can be heard to 0 mmHg. Under these circumstances, the BP measurement should be repeated with less pressure on the head of the stethoscope.<sup>4</sup> Only if the very low K5 persists should K4 (muffling of the sounds) be recorded as the DBP.

The standard device for BP measurements has been the mercury manometer.<sup>14</sup> Because of its environmental toxicity, mercury has been increasingly removed from health care settings. Aneroid manometers are quite accurate when calibrated on a semiannual basis<sup>15</sup> and are recommended when mercury-column devices cannot be obtained.

Auscultation remains the recommended method of BP measurement in children, under most circumstances. Oscillometric devices measure mean arterial BP and then calculate systolic and diastolic values.<sup>16</sup> The algorithms used by companies are proprietary and differ from company to company and device to device. These devices can yield results that

vary widely when one is compared with another,<sup>17</sup> and they do not always closely match BP values obtained by auscultation.<sup>18</sup> Oscillometric devices must be validated on a regular basis. Protocols for validation have been developed,<sup>19,20</sup> but the validation process is very difficult.

Two advantages of automatic devices are their ease of use and the minimization of observer bias or digit preference.<sup>16</sup> Use of the automated devices is preferred for BP measurement in newborns and young infants, in whom auscultation is difficult, and in the intensive care setting where frequent BP measurement is needed. An elevated BP reading obtained with an oscillometric device should be repeated using auscultation.

*Elevated BP must be confirmed on repeated visits before characterizing a child as having hypertension.* Confirming an elevated BP measurement is important, because BP at high levels tends to fall on subsequent measurement as the result of (1) an accommodation effect (i.e., reduction of anxiety by the patient from one visit to the next), and (2) regression to the mean. BP level is not static but varies even under standard resting conditions. Therefore, except in the presence of severe hypertension, a more precise characterization of a person's BP level is an average of multiple BP measurements taken over weeks to months.

## **AMBULATORY BLOOD PRESSURE MONITORING**

Ambulatory BP monitoring (ABPM) refers to a procedure in which a portable BP device, worn by the patient, records BP over a specified period, usually 24 hours. ABPM is very useful in the evaluation of hypertension in children.<sup>21-23</sup> By frequent measurement and recording of BP, ABPM enables computation of the mean BP during the day, night, and over 24 hours as well as various measures to determine the degree to which BP exceeds the upper limit of normal over a given time period (i.e., the BP load). ABPM is especially helpful in the evaluation of white-coat hypertension, as well as the risk for hypertensive organ injury, apparent drug resistance, and hypotensive symptoms with antihypertensive drugs. ABPM is also useful for evaluating patients for whom more information on BP patterns is needed, such as those with episodic hypertension, chronic kidney disease, diabetes, and autonomic dysfunction. Conducting ABPM requires specific equipment and trained staff. Therefore, ABPM in children and adolescents should be used by experts in the field of pediatric hypertension who are experienced in its use and interpretation.

# Blood Pressure Tables

- *BP standards based on sex, age, and height provide a precise classification of BP according to body size.*
- *The revised BP tables now include the 50th, 90th, 95th, and 99th percentiles (with standard deviations) by sex, age, and height.*

In children and adolescents, the normal range of BP is determined by body size and age. BP standards that are based on sex, age, and height provide a more precise classification of BP according to body size. This approach avoids misclassifying children who are very tall or very short.

The BP tables are revised to include the new height percentile data ([www.cdc.gov/growthcharts/](http://www.cdc.gov/growthcharts/))<sup>24</sup> as well as the addition of BP data from the NHANES 1999–2000. Demographic information on the source of the BP data is provided in appendix A. The 50th, 90th, 95th, and 99th percentiles of SBP and DBP (using K5) for height by sex and age are given for boys and girls in tables 3 and 4. Although new data have been added, the sex, age, and height BP levels for the 90th and 95th percentiles have changed minimally from the last report. The 50th percentile has been added to the tables to provide the clinician with the BP level at the midpoint of the normal range. Although the 95th percentile provides a BP level that defines hypertension, management decisions about children with hypertension should be determined by the degree or severity of hypertension. Therefore, the 99th percentile has been added to facilitate clinical decisionmaking in the plan for evaluation. Standards for SBP and DBP for infants younger than 1 year are available.<sup>25</sup> In children younger than 1 year, SBP has been used to define hypertension.

To use the tables in a clinical setting, the height percentile is determined by using the

newly revised CDC Growth Charts ([www.cdc.gov/growthcharts/](http://www.cdc.gov/growthcharts/)). The child's measured SBP and DBP are compared with the numbers provided in the table (boys or girls) according to the child's age and height percentile. The child is normotensive if the BP is below the 90th percentile. If the BP is equal to or above the 90th percentile, the BP measurement should be repeated at that visit to verify an elevated BP. BP measurements between the 90th and 95th percentiles indicate prehypertension and warrant reassessment and consideration of other risk factors. (See table 5.) In addition, if an adolescent's BP is greater than 120/80 mmHg, the patient should be considered to be prehypertensive even if this value is less than the 90th percentile. This BP level typically occurs for SBP at age 12 years and for DBP at age 16 years.

If the child's BP (systolic or diastolic) is at or above the 95th percentile, the child may be hypertensive, and the measurement must be repeated on at least two additional occasions to confirm the diagnosis. Staging of BP, according to the extent to which a child's BP exceeds the 95th percentile, is helpful in developing a management plan for evaluation and treatment that is most appropriate for an individual patient. On repeated measurement, hypertensive children may have BP levels that are only a few mmHg above the 95th percentile; these children would be managed differently from hypertensive children who have BP levels that are 15–20 mmHg above the 95th percentile. An important clinical decision is to determine which hypertensive

children require more immediate attention for elevated BP. The difference between the 95th and 99th percentiles is only 7–10 mmHg and is not large enough, particularly in view of the variability in BP measurements, to adequately distinguish mild hypertension—where limited evaluation is most appropriate—from more severe hypertension where more immediate and extensive intervention is indicated.

Therefore, Stage 1 hypertension is the designation for BP levels that range from the 95th percentile to 5 mmHg above the 99th percentile. Stage 2 hypertension is the designation for BP levels that are higher than 5 mmHg above the 99th percentile. Once confirmed on repeated measures, Stage 1 hypertension allows time for evaluation before initiating treatment unless the patient is symptomatic. Patients with Stage 2 hypertension may need more prompt evaluation and pharmacologic therapy. Symptomatic patients with Stage 2 hypertension require immediate treatment and consultation with experts in pediatric hypertension. These categories are parallel to the staging of hypertension in adults, as noted in JNC 7.<sup>2</sup>

### USING THE BLOOD PRESSURE TABLES

1. Use the standard height charts to determine the height percentile.
2. Measure and record the child's SBP and DBP.
3. Use the correct gender table for SBP and DBP.
4. Find the child's age on the left side of the table. Follow the age row horizontally across the table to the intersection of the line for the height percentile (vertical column).

5. There, find the 50th, 90th, 95th, and 99th percentiles for SBP in the left columns and for DBP in the right columns.
  - BP less than the 90th percentile is normal.
  - BP between the 90th and 95th percentile is prehypertension. In adolescents, BP equal to or exceeding 120/80 mmHg is prehypertension, even if this figure is less than the 90th percentile.
  - BP greater than the 95th percentile may be hypertension.
6. If the BP is greater than the 90th percentile, the BP should be repeated twice at the same office visit, and an average SBP and DBP should be used.
7. If the BP is greater than the 95th percentile, BP should be staged. If Stage 1 (95th percentile to the 99th percentile plus 5 mmHg), BP measurements should be repeated on two more occasions. If hypertension is confirmed, evaluation should proceed as described in table 7. If BP is Stage 2 (>99th percentile plus 5 mmHg), prompt referral should be made for evaluation and therapy. If the patient is symptomatic, immediate referral and treatment are indicated. Those patients with a compelling indication, as noted in table 6, would be treated as the next higher category of hypertension.

TABLE 3

## Blood Pressure Levels for Boys by Age and Height Percentile\*

Age (Year)	BP Percentile ↓	Systolic BP (mmHg)							Diastolic BP (mmHg)						
		← Percentile of Height →							← Percentile of Height →						
		5th	10th	25th	50th	75th	90th	95th	5th	10th	25th	50th	75th	90th	95th
1	50th	80	81	83	85	87	88	89	34	35	36	37	38	39	39
	90th	94	95	97	99	100	102	103	49	50	51	52	53	53	54
	95th	98	99	101	103	104	106	106	54	54	55	56	57	58	58
	99th	105	106	108	110	112	113	114	61	62	63	64	65	66	66
2	50th	84	85	87	88	90	92	92	39	40	41	42	43	44	44
	90th	97	99	100	102	104	105	106	54	55	56	57	58	58	59
	95th	101	102	104	106	108	109	110	59	59	60	61	62	63	63
	99th	109	110	111	113	115	117	117	66	67	68	69	70	71	71
3	50th	86	87	89	91	93	94	95	44	44	45	46	47	48	48
	90th	100	101	103	105	107	108	109	59	59	60	61	62	63	63
	95th	104	105	107	109	110	112	113	63	63	64	65	66	67	67
	99th	111	112	114	116	118	119	120	71	71	72	73	74	75	75
4	50th	88	89	91	93	95	96	97	47	48	49	50	51	51	52
	90th	102	103	105	107	109	110	111	62	63	64	65	66	66	67
	95th	106	107	109	111	112	114	115	66	67	68	69	70	71	71
	99th	113	114	116	118	120	121	122	74	75	76	77	78	78	79
5	50th	90	91	93	95	96	98	98	50	51	52	53	54	55	55
	90th	104	105	106	108	110	111	112	65	66	67	68	69	69	70
	95th	108	109	110	112	114	115	116	69	70	71	72	73	74	74
	99th	115	116	118	120	121	123	123	77	78	79	80	81	81	82
6	50th	91	92	94	96	98	99	100	53	53	54	55	56	57	57
	90th	105	106	108	110	111	113	113	68	68	69	70	71	72	72
	95th	109	110	112	114	115	117	117	72	72	73	74	75	76	76
	99th	116	117	119	121	123	124	125	80	80	81	82	83	84	84
7	50th	92	94	95	97	99	100	101	55	55	56	57	58	59	59
	90th	106	107	109	111	113	114	115	70	70	71	72	73	74	74
	95th	110	111	113	115	117	118	119	74	74	75	76	77	78	78
	99th	117	118	120	122	124	125	126	82	82	83	84	85	86	86
8	50th	94	95	97	99	100	102	102	56	57	58	59	60	60	61
	90th	107	109	110	112	114	115	116	71	72	72	73	74	75	76
	95th	111	112	114	116	118	119	120	75	76	77	78	79	79	80
	99th	119	120	122	123	125	127	127	83	84	85	86	87	87	88
9	50th	95	96	98	100	102	103	104	57	58	59	60	61	61	62
	90th	109	110	112	114	115	117	118	72	73	74	75	76	76	77
	95th	113	114	116	118	119	121	121	76	77	78	79	80	81	81
	99th	120	121	123	125	127	128	129	84	85	86	87	88	88	89
10	50th	97	98	100	102	103	105	106	58	59	60	61	61	62	63
	90th	111	112	114	115	117	119	119	73	73	74	75	76	77	78
	95th	115	116	117	119	121	122	123	77	78	79	80	81	81	82
	99th	122	123	125	127	128	130	130	85	86	86	88	88	89	90

Age (Year)	BP Percentile ↓	Systolic BP (mmHg)							Diastolic BP (mmHg)						
		← Percentile of Height →							← Percentile of Height →						
		5th	10th	25th	50th	75th	90th	95th	5th	10th	25th	50th	75th	90th	95th
11	50th	99	100	102	104	105	107	107	59	59	60	61	62	63	63
	90th	113	114	115	117	119	120	121	74	74	75	76	77	78	78
	95th	117	118	119	121	123	124	125	78	78	79	80	81	82	82
	99th	124	125	127	129	130	132	132	86	86	87	88	89	90	90
12	50th	101	102	104	106	108	109	110	59	60	61	62	63	63	64
	90th	115	116	118	120	121	123	123	74	75	75	76	77	78	79
	95th	119	120	122	123	125	127	127	78	79	80	81	82	82	83
	99th	126	127	129	131	133	134	135	86	87	88	89	90	90	91
13	50th	104	105	106	108	110	111	112	60	60	61	62	63	64	64
	90th	117	118	120	122	124	125	126	75	75	76	77	78	79	79
	95th	121	122	124	126	128	129	130	79	79	80	81	82	83	83
	99th	128	130	131	133	135	136	137	87	87	88	89	90	91	91
14	50th	106	107	109	111	113	114	115	60	61	62	63	64	65	65
	90th	120	121	123	125	126	128	128	75	76	77	78	79	79	80
	95th	124	125	127	128	130	132	132	80	80	81	82	83	84	84
	99th	131	132	134	136	138	139	140	87	88	89	90	91	92	92
15	50th	109	110	112	113	115	117	117	61	62	63	64	65	66	66
	90th	122	124	125	127	129	130	131	76	77	78	79	80	80	81
	95th	126	127	129	131	133	134	135	81	81	82	83	84	85	85
	99th	134	135	136	138	140	142	142	88	89	90	91	92	93	93
16	50th	111	112	114	116	118	119	120	63	63	64	65	66	67	67
	90th	125	126	128	130	131	133	134	78	78	79	80	81	82	82
	95th	129	130	132	134	135	137	137	82	83	83	84	85	86	87
	99th	136	137	139	141	143	144	145	90	90	91	92	93	94	94
17	50th	114	115	116	118	120	121	122	65	66	66	67	68	69	70
	90th	127	128	130	132	134	135	136	80	80	81	82	83	84	84
	95th	131	132	134	136	138	139	140	84	85	86	87	87	88	89
	99th	139	140	141	143	145	146	147	92	93	93	94	95	96	97

BP, blood pressure

\* The 90th percentile is 1.28 SD, 95th percentile is 1.645 SD, and the 99th percentile is 2.326 SD over the mean. For research purposes, the standard deviations in appendix table B-1 allow one to compute BP Z-scores and percentiles for boys with height percentiles given in table 3 (i.e., the 5th, 10th, 25th, 50th, 75th, 90th, and 95th percentiles). These height percentiles must be converted to height Z-scores given by (5% = -1.645; 10% = -1.28; 25% = -0.68; 50% = 0; 75% = 0.68; 90% = 1.28; 95% = 1.645) and then computed according to the methodology in steps 2-4 described in appendix B. For children with height percentiles other than these, follow steps 1-4 as described in appendix B.

TABLE 4

## Blood Pressure Levels for Girls by Age and Height Percentile\*

Age (Year)	BP Percentile ↓	Systolic BP (mmHg)							Diastolic BP (mmHg)						
		← Percentile of Height →							← Percentile of Height →						
		5th	10th	25th	50th	75th	90th	95th	5th	10th	25th	50th	75th	90th	95th
1	50th	83	84	85	86	88	89	90	38	39	39	40	41	41	42
	90th	97	97	98	100	101	102	103	52	53	53	54	55	55	56
	95th	100	101	102	104	105	106	107	56	57	57	58	59	59	60
	99th	108	108	109	111	112	113	114	64	64	65	65	66	67	67
2	50th	85	85	87	88	89	91	91	43	44	44	45	46	46	47
	90th	98	99	100	101	103	104	105	57	58	58	59	60	61	61
	95th	102	103	104	105	107	108	109	61	62	62	63	64	65	65
	99th	109	110	111	112	114	115	116	69	69	70	70	71	72	72
3	50th	86	87	88	89	91	92	93	47	48	48	49	50	50	51
	90th	100	100	102	103	104	106	106	61	62	62	63	64	64	65
	95th	104	104	105	107	108	109	110	65	66	66	67	68	68	69
	99th	111	111	113	114	115	116	117	73	73	74	74	75	76	76
4	50th	88	88	90	91	92	94	94	50	50	51	52	52	53	54
	90th	101	102	103	104	106	107	108	64	64	65	66	67	67	68
	95th	105	106	107	108	110	111	112	68	68	69	70	71	71	72
	99th	112	113	114	115	117	118	119	76	76	76	77	78	79	79
5	50th	89	90	91	93	94	95	96	52	53	53	54	55	55	56
	90th	103	103	105	106	107	109	109	66	67	67	68	69	69	70
	95th	107	107	108	110	111	112	113	70	71	71	72	73	73	74
	99th	114	114	116	117	118	120	120	78	78	79	79	80	81	81
6	50th	91	92	93	94	96	97	98	54	54	55	56	56	57	58
	90th	104	105	106	108	109	110	111	68	68	69	70	70	71	72
	95th	108	109	110	111	113	114	115	72	72	73	74	74	75	76
	99th	115	116	117	119	120	121	122	80	80	80	81	82	83	83
7	50th	93	93	95	96	97	99	99	55	56	56	57	58	58	59
	90th	106	107	108	109	111	112	113	69	70	70	71	72	72	73
	95th	110	111	112	113	115	116	116	73	74	74	75	76	76	77
	99th	117	118	119	120	122	123	124	81	81	82	82	83	84	84
8	50th	95	95	96	98	99	100	101	57	57	57	58	59	60	60
	90th	108	109	110	111	113	114	114	71	71	71	72	73	74	74
	95th	112	112	114	115	116	118	118	75	75	75	76	77	78	78
	99th	119	120	121	122	123	125	125	82	82	83	83	84	85	86
9	50th	96	97	98	100	101	102	103	58	58	58	59	60	61	61
	90th	110	110	112	113	114	116	116	72	72	72	73	74	75	75
	95th	114	114	115	117	118	119	120	76	76	76	77	78	79	79
	99th	121	121	123	124	125	127	127	83	83	84	84	85	86	87
10	50th	98	99	100	102	103	104	105	59	59	59	60	61	62	62
	90th	112	112	114	115	116	118	118	73	73	73	74	75	76	76
	95th	116	116	117	119	120	121	122	77	77	77	78	79	80	80
	99th	123	123	125	126	127	129	129	84	84	85	86	86	87	88

Age (Year)	BP Percentile ↓	Systolic BP (mmHg)							Diastolic BP (mmHg)						
		← Percentile of Height →							← Percentile of Height →						
		5th	10th	25th	50th	75th	90th	95th	5th	10th	25th	50th	75th	90th	95th
11	50th	100	101	102	103	105	106	107	60	60	60	61	62	63	63
	90th	114	114	116	117	118	119	120	74	74	74	75	76	77	77
	95th	118	118	119	121	122	123	124	78	78	78	79	80	81	81
	99th	125	125	126	128	129	130	131	85	85	86	87	87	88	89
12	50th	102	103	104	105	107	108	109	61	61	61	62	63	64	64
	90th	116	116	117	119	120	121	122	75	75	75	76	77	78	78
	95th	119	120	121	123	124	125	126	79	79	79	80	81	82	82
	99th	127	127	128	130	131	132	133	86	86	87	88	88	89	90
13	50th	104	105	106	107	109	110	110	62	62	62	63	64	65	65
	90th	117	118	119	121	122	123	124	76	76	76	77	78	79	79
	95th	121	122	123	124	126	127	128	80	80	80	81	82	83	83
	99th	128	129	130	132	133	134	135	87	87	88	89	89	90	91
14	50th	106	106	107	109	110	111	112	63	63	63	64	65	66	66
	90th	119	120	121	122	124	125	125	77	77	77	78	79	80	80
	95th	123	123	125	126	127	129	129	81	81	81	82	83	84	84
	99th	130	131	132	133	135	136	136	88	88	89	90	90	91	92
15	50th	107	108	109	110	111	113	113	64	64	64	65	66	67	67
	90th	120	121	122	123	125	126	127	78	78	78	79	80	81	81
	95th	124	125	126	127	129	130	131	82	82	82	83	84	85	85
	99th	131	132	133	134	136	137	138	89	89	90	91	91	92	93
16	50th	108	108	110	111	112	114	114	64	64	65	66	66	67	68
	90th	121	122	123	124	126	127	128	78	78	79	80	81	81	82
	95th	125	126	127	128	130	131	132	82	82	83	84	85	85	86
	99th	132	133	134	135	137	138	139	90	90	90	91	92	93	93
17	50th	108	109	110	111	113	114	115	64	65	65	66	67	67	68
	90th	122	122	123	125	126	127	128	78	79	79	80	81	81	82
	95th	125	126	127	129	130	131	132	82	83	83	84	85	85	86
	99th	133	133	134	136	137	138	139	90	90	91	91	92	93	93

BP, blood pressure

\* The 90th percentile is 1.28 SD, 95th percentile is 1.645 SD, and the 99th percentile is 2.326 SD over the mean. For research purposes, the standard deviations in appendix table B-1 allow one to compute BP Z-scores and percentiles for girls with height percentiles given in table 4 (i.e., the 5th, 10th, 25th, 50th, 75th, 90th, and 95th percentiles). These height percentiles must be converted to height Z-scores given by (5% = -1.645; 10% = -1.28; 25% = -0.68; 50% = 0; 75% = 0.68; 90% = 1.28; 95% = 1.645) and then computed according to the methodology in steps 2-4 described in appendix B. For children with height percentiles other than these, follow steps 1-4 as described in appendix B.

TABLE 5

### Classification of Hypertension in Children and Adolescents, With Measurement Frequency and Therapy Recommendations

	SBP or DBP Percentile*	Frequency of BP Measurement	Therapeutic Lifestyle Changes	Pharmacologic Therapy
Normal	<90th	Recheck at next scheduled physical examination.	Encourage healthy diet, sleep, and physical activity.	—
Prehypertension	90th to <95th or if BP exceeds 120/80 mmHg even if below 90th percentile up to <95th percentile <sup>†</sup>	Recheck in 6 months.	Weight-management counseling if overweight, introduce physical activity and diet management. <sup>‡</sup>	None unless compelling indications such as CKD, diabetes mellitus, heart failure, or LVH exist
Stage 1 hypertension	95th percentile to the 99th percentile plus 5 mmHg	Recheck in 1–2 weeks or sooner if the patient is symptomatic; if persistently elevated on two additional occasions, evaluate or refer to source of care within 1 month.	Weight-management counseling if overweight, introduce physical activity and diet management. <sup>‡</sup>	Initiate therapy based on indications in Table 6 or if compelling indications as above.
Stage 2 hypertension	>99th percentile plus 5 mmHg	Evaluate or refer to source of care within 1 week or immediately if the patient is symptomatic.	Weight-management counseling if overweight, introduce physical activity and diet management. <sup>‡</sup>	Initiate therapy. <sup>§</sup>

BP, blood pressure; CKD, chronic kidney disease; DBP, diastolic blood pressure; LVH, left ventricular hypertrophy; SBP, systolic blood pressure

\* For sex, age, and height measured on at least three separate occasions; if systolic and diastolic categories are different, categorize by the higher value.

<sup>†</sup> This occurs typically at 12 years old for SBP and at 16 years old for DBP.

<sup>‡</sup> Parents and children trying to modify the eating plan to the Dietary Approaches to Stop Hypertension (DASH) eating plan could benefit from consultation with a registered or licensed nutritionist to get them started.

<sup>§</sup> More than one drug may be required.

TABLE 6

### Indications for Antihypertensive Drug Therapy in Children

- Symptomatic hypertension
- Secondary hypertension
- Hypertensive target-organ damage
- Diabetes (types 1 and 2)
- Persistent hypertension despite nonpharmacologic measures

TABLE 7

## Clinical Evaluation of Confirmed Hypertension

Study or Procedure	Purpose	Target Population
<b>Evaluation for identifiable causes</b>		
History, including sleep history, family history, risk factors, diet, and habits such as smoking and drinking alcohol; physical examination	History and physical examination help focus subsequent evaluation	All children with persistent BP $\geq$ 95th percentile
BUN, creatinine, electrolytes, urinalysis, and urine culture	R/O renal disease and chronic pyelonephritis	All children with persistent BP $\geq$ 95th percentile
CBC	R/O anemia, consistent with chronic renal disease	All children with persistent BP $\geq$ 95th percentile
Renal U/S	R/O renal scar, congenital anomaly, or disparate renal size	All children with persistent BP $\geq$ 95th percentile
<b>Evaluation for comorbidity</b>		
Fasting lipid panel, fasting glucose	Identify hyperlipidemia, identify metabolic abnormalities	Overweight patients with BP at 90th–94th percentile; all patients with BP $\geq$ 95th percentile. Family history of hypertension or cardiovascular disease. Child with chronic renal disease
Drug screen	Identify substances that might cause hypertension	History suggestive of possible contribution by substances or drugs
Polysomnography	Identify sleep disorder in association with hypertension	History of loud, frequent snoring
<b>Evaluation for target-organ damage</b>		
Echocardiogram	Identify LVH and other indications of cardiac involvement	Patients with comorbid risk factors* and BP 90th–94th percentile; all patients with BP $\geq$ 95th percentile
Retinal exam	Identify retinal vascular changes	Patients with comorbid risk factors* and BP 90th–94th percentile; all patients with BP $\geq$ 95th percentile
<b>Further evaluation as indicated</b>		
Ambulatory BP monitoring	Identify white-coat hypertension, abnormal diurnal BP pattern, BP load	Patients in whom white-coat hypertension is suspected, and when other information on BP pattern is needed
Plasma renin determination	Identify low renin, suggesting mineralo-corticoid-related disease	Young children with Stage 1 hypertension and any child or adolescent with Stage 2 hypertension  Positive family history of severe hypertension
Renovascular imaging <ul style="list-style-type: none"> <li>• Isotopic scintigraphy (renal scan)</li> <li>• Magnetic resonance angiography</li> <li>• Duplex Doppler flow studies</li> <li>• 3-Dimensional CT</li> <li>• Arteriography: DSA or classic</li> </ul>	Identify renovascular disease	Young children with Stage 1 hypertension and any child or adolescent with Stage 2 hypertension
Plasma and urine steroid levels	Identify steroid-mediated hypertension	Young children with Stage 1 hypertension and any child or adolescent with Stage 2 hypertension
Plasma and urine catecholamines	Identify catecholamine-mediated hypertension	Young children with Stage 1 hypertension and any child or adolescent with Stage 2 hypertension

BP, blood pressure; BUN, blood urea nitrogen; CBC, complete blood count; CT, computerized tomography; DSA, digital subtraction angiography;

LVH, left ventricular hypertrophy; R/O, rule out; U/S, ultrasound

\* Comorbid risk factors also include diabetes mellitus and kidney disease.

# Primary Hypertension and Evaluation for Comorbidities

- *Primary hypertension is identifiable in children and adolescents.*
- *Both hypertension and prehypertension have become a significant health issue in the young due to the strong association of high BP with overweight and the marked increase in the prevalence of overweight children.*
- *The evaluation of hypertensive children should include assessment for additional risk factors.*
- *Due to an association of sleep apnea with overweight and high BP, a sleep history should be obtained.*

High BP in childhood had been considered a risk factor for hypertension in early adulthood. However, primary (essential) hypertension is now identifiable in children and adolescents. Primary hypertension in childhood is usually characterized by mild or Stage 1 hypertension and is often associated with a positive family history of hypertension or cardiovascular disease (CVD). Children and adolescents with primary hypertension are frequently overweight. Data on healthy adolescents obtained in school health-screening programs demonstrate that the prevalence of hypertension increases progressively with increasing body mass index (BMI), and hypertension is detectable in approximately 30 percent of overweight children (BMI >95th percentile).<sup>26</sup> The strong association of high BP with obesity and the marked increase in the prevalence of childhood obesity<sup>27</sup> indicate that both hypertension and prehypertension are becoming a significant health issue in the young. Overweight children frequently have some degree of insulin resistance—a prediabetic condition. Overweight and high BP are also components of the insulin-resistance syndrome or metabolic syndrome, a condition of multiple metabolic risk factors for CVD as well as for type 2 diabetes.<sup>28,29</sup> The clustering of other CVD risk factors that are included in the insulin-resistance syndrome (high triglycerides, low

high-density lipoprotein cholesterol [HDL-C], truncal obesity, hyperinsulinemia) is significantly greater among children with high BP than in children with normal BP.<sup>30</sup> Recent reports from studies that examined childhood data estimate that the insulin-resistance syndrome is present in 30 percent of overweight children with BMI greater than the 95th percentile.<sup>31</sup> Historically, hypertension in childhood was considered a simple independent risk factor for CVD, but its link to the other risk factors in the insulin-resistance syndrome indicates that a broader approach is more appropriate in affected children.

Primary hypertension often clusters with other risk factors.<sup>31,32</sup> Therefore, the medical history, physical examination, and laboratory evaluation of hypertensive children and adolescents should include a comprehensive assessment for additional cardiovascular risk. These risk factors, in addition to high BP and overweight, include low plasma HDL-C, elevated plasma triglyceride, and abnormal glucose tolerance. Fasting plasma insulin concentration is generally elevated, but an elevated insulin concentration may be reflective only of obesity and is not diagnostic of the insulin-resistance syndrome. To identify other cardiovascular risk factors, a fasting lipid panel and fasting glucose level should be obtained in children who are overweight and

have BP between the 90th and 94th percentile and in all children with BP greater than the 95th percentile. If there is a strong family history of type 2 diabetes, a hemoglobin A1c or glucose tolerance test may also be considered. These metabolic risk factors should be repeated periodically to detect changes in the level of cardiovascular risk over time. Fewer data are available on the utility of other tests in children (e.g., plasma uric acid, or homocysteine and lp(a) levels), and the use of these measures should depend on family history.

Sleep disorders, including sleep apnea, are associated with hypertension, coronary artery disease, heart failure, and stroke in adults.<sup>33,34</sup> Although limited data are available, they suggest an association of sleep-disordered breathing and higher BP in children.<sup>35,36</sup>

Approximately 15 percent of children snore, and at least 1–3 percent have sleep-disordered breathing.<sup>35</sup> Because of the associations with hypertension and the frequency of occurrence of sleep disorders, particularly among overweight children, a history of sleeping patterns should be obtained in a child with hypertension. One practical strategy for

identifying children with a sleep problem or sleep disorder is to obtain a brief sleep history, using an instrument called BEARS.<sup>37</sup>(table 1.1) BEARS addresses five major sleep domains that provide a simple but comprehensive screen for the major sleep disorders affecting children ages 2–18. The components of BEARS include: **B**edtime problems, **E**xcessive daytime sleepiness, **A**wakenings during the night, **R**egularity and duration of sleep, and **S**leep-disordered breathing (snoring). Each of these domains has an age-appropriate trigger question and includes responses of both parent and child, as appropriate. This brief screening for sleep history can be completed in about 5 minutes.

In a child with primary hypertension, the presence of any comorbidity that is associated with hypertension carries the potential to increase the risk for CVD and can have an adverse effect on health outcome. Consideration of these associated risk factors and appropriate evaluation in those children in whom the hypertension is verified are important in planning and implementing therapies that reduce the comorbidity risk as well as control BP.

# Evaluation for Secondary Hypertension

- *Secondary hypertension is more common in children than in adults.*
- *Because overweight is strongly linked to hypertension, BMI should be calculated as part of the physical examination.*
- *Once hypertension is confirmed, BP should be measured in both arms and in a leg.*
- *Very young children, children with Stage 2 hypertension, and children or adolescents with clinical signs that suggest systemic conditions associated with hypertension should be evaluated more completely than in those with Stage 1 hypertension.*

Secondary hypertension is more common in children than in adults. The possibility that some underlying disorder may be the cause of the hypertension should be considered in every child or adolescent who has elevated BP. However, the extent of an evaluation for detection of a possible underlying cause should be individualized for each child. Very young children, children with Stage 2 hypertension, and children or adolescents with clinical signs that suggest the presence of systemic conditions associated with hypertension should be evaluated more extensively as compared to those with Stage 1 hypertension.<sup>38</sup> Present technologies may facilitate less invasive evaluation than in the past, although experience in using newer modalities with children is still limited.

A thorough history and physical examination are the first steps in the evaluation of any child with persistently elevated BP. Elicited information should aim to identify not only signs and symptoms due to high BP but also clinical findings that might uncover an underlying systemic disorder. Thus, it is important to seek signs and symptoms suggesting renal disease (gross hematuria, edema, fatigue), heart disease (chest pain, exertional dyspnea, palpitations), and diseases of other organ systems (e.g., endocrinologic, rheumatologic). Past medical history should elicit information to focus the subsequent evaluation and to

uncover definable causes of hypertension. Questions should be asked about prior hospitalizations, trauma, urinary tract infections, snoring and other sleep problems. Questions should address family history of hypertension, diabetes, obesity, sleep apnea, renal disease, other CVD (hyperlipidemia, stroke), and familial endocrinopathies. Many drugs can increase BP, so it is important to inquire directly about use of over-the-counter, prescription, and illicit drugs. Equally important are specific questions aimed at identifying the use of nutritional supplements, especially preparations aimed at enhancing athletic performance.

## PHYSICAL EXAMINATION

The child's height, weight, and percentiles for age should be determined at the start of the physical examination. Because obesity is strongly linked to hypertension, BMI should be calculated from the height and weight, and the BMI percentile should be calculated. Poor growth may indicate an underlying chronic illness. When hypertension is confirmed, BP should be measured in both arms and in a leg. Normally, BP is 10–20 mmHg higher in the legs than the arms. If the leg BP is lower than the arm BP, or if femoral pulses are weak or absent, coarctation of the aorta may be present. Obesity alone is an insufficient explanation for diminished femoral pulses in the

presence of high BP. The remainder of the physical examination should pursue clues found on history and should focus on findings that may indicate the cause and severity of hypertension. Table 8 lists important physical exam findings in hypertensive children.<sup>39</sup>

The physical examination in hypertensive children is frequently normal except for the BP elevation. The extent of the laboratory evaluation is based on the child's age, history, physical examination findings, and level of BP elevation. The majority of children with secondary hypertension will have renal or renovascular causes for the BP elevation. Therefore, screening tests are designed to have a high likelihood of detecting children and adolescents who are so affected. These tests are easily obtained in most primary care offices and community hospitals. Additional evaluation must be tailored to the specific child and situation. The risk factors, or comorbid conditions, associated with primary hypertension should be included in the evaluation of hypertension in all children, as well as efforts to determine any evidence of target-organ damage.

### **ADDITIONAL DIAGNOSTIC STUDIES FOR HYPERTENSION**

Additional diagnostic studies may be appropriate in the evaluation of hypertension in a child or adolescent, particularly if there is a high degree of suspicion that an underlying disorder is present. Such procedures are listed in table 7. ABPM, discussed previously, has application in evaluating both primary and secondary hypertension. ABPM is also used to detect white-coat hypertension.

### **RENIN PROFILING**

Plasma renin level or plasma renin activity (PRA) is a useful screening test for mineralocorticoid-related diseases. With these disorders, the PRA is very low or unmeasurable by the laboratory and may be associated with relative hypokalemia. PRA levels are higher in patients who have renal artery stenosis. However, approximately 15 percent

of children with arteriographically evident renal artery stenosis have normal PRA values.<sup>40–42</sup> Assays for direct measurement of renin, a different technique than PRA, are commonly used, although extensive normative data in children and adolescents are unavailable.

### **EVALUATION FOR POSSIBLE RENOVASCULAR HYPERTENSION**

Renovascular hypertension is a consequence of an arterial lesion or lesions impeding blood flow to one or both kidneys or to one or more intrarenal segments.<sup>43,44</sup> Affected children usually, but not invariably, have markedly elevated BP.<sup>40,44</sup> Evaluation for renovascular disease also should be considered in infants or children with other known predisposing factors, such as prior umbilical artery catheter placements or neurofibromatosis.<sup>44,45</sup> A number of newer diagnostic techniques are presently available for evaluation of renovascular disease, but experience in their use in pediatric patients is limited. Consequently, the recommended approaches generally use older techniques, such as standard intra-arterial angiography, digital-subtraction angiography (DSA), and scintigraphy (with or without angiotensin-converting enzyme [ACE] inhibition).<sup>44</sup> As technologies evolve, children should be referred for imaging studies to centers that have expertise in the radiological evaluation of childhood hypertension.

### **INVASIVE STUDIES**

Intra-arterial DSA with contrast is used more frequently than standard angiography, but, because of intra-arterial injection, this method remains invasive. DSA also can be accomplished by using a rapid injection of contrast into a peripheral vein, but quality of views and the size of pediatric veins make this technique useful only for older children. DSA and formal arteriography are still considered the “gold standard,” but these studies should be undertaken only when surgical or invasive interventional radiologic techniques are being contemplated for anatomic correction.<sup>46</sup>

TABLE 8

## Examples of Physical Examination Findings Suggestive of Definable Hypertension\*

	Finding†	Possible Etiology
Vital signs	Tachycardia	Hyperthyroidism, pheochromocytoma, neuroblastoma, primary hypertension
	Decreased lower extremity pulses; drop in BP from upper to lower extremities	Coarctation of the aorta
Eyes	Retinal changes	Severe hypertension, more likely to be associated with secondary hypertension
Ear, nose, and throat (ENT)	Adenotonsillar hypertrophy	Suggests association with sleep-disordered breathing (sleep apnea), snoring
Height/weight	Growth retardation	Chronic renal failure
	Obesity (high BMI)	Primary hypertension
	Truncal obesity	Cushing syndrome, insulin resistance syndrome
Head and neck	Moon facies	Cushing syndrome
	Elfin facies	Williams syndrome
	Webbed neck	Turner syndrome
	Thyromegaly	Hyperthyroidism
Skin	Pallor, flushing, diaphoresis	Pheochromocytoma
	Acne, hirsutism, striae	Cushing syndrome, anabolic steroid abuse
	Café-au-lait spots	Neurofibromatosis
	Adenoma sebaceum	Tuberous sclerosis
	Malar rash	Systemic lupus erythematosus
	Acanthosis nigricans	Type 2 diabetes
Chest	Widely spaced nipples	Turner syndrome
	Heart murmur	Coarctation of the aorta
	Friction rub	Systemic lupus erythematosus (pericarditis), collagen-vascular disease, end stage renal disease with uremia
	Apical heave	Left ventricular hypertrophy/chronic hypertension
Abdomen	Mass	Wilms tumor, neuroblastoma, pheochromocytoma
	Epigastric/flank bruit	Renal artery stenosis
	Palpable kidneys	Polycystic kidney disease, hydronephrosis, multicystic-dysplastic kidney, mass (see above)
Genitalia	Ambiguous/virilization	Adrenal hyperplasia
Extremities	Joint swelling	Systemic lupus erythematosus, collagen vascular disease
	Muscle weakness	Hyperaldosteronism, Liddle syndrome

BMI, body mass index; BP, blood pressure

\* Adapted from Flynn, JT. Evaluation and management of hypertension in childhood. *Prog Pediatr Cardiol* 2001;12:177–88.

† Findings listed are examples of physical findings and do not represent all possible physical findings.

Newer imaging techniques may be used in children with vascular lesions. Magnetic resonance angiography (MRA) is increasingly feasible for the evaluation of pediatric renovascular disease, but it is still best for detecting abnormalities in the main renal artery and its primary branches.<sup>47-49</sup> Imaging with magnetic resonance requires that the patient be relatively immobile for extended

periods—a significant difficulty for small children. At present, studies are needed to assess the effectiveness of MRA in the diagnosis of children with renovascular disease. Newer methods, including 3-dimensional (3-D) reconstructions of computerized tomography (CT) images, or spiral CT with contrast, appear promising in evaluating children who may have renovascular disease.<sup>50</sup>

# Target-Organ Abnormalities in Childhood Hypertension

- *Target-organ abnormalities are commonly associated with hypertension in children and adolescents.*
- *Left ventricular hypertrophy (LVH) is the most prominent evidence of target-organ damage.*
- *Pediatric patients with established hypertension should have echocardiographic assessment of left ventricular mass at diagnosis and periodically thereafter.*
- *The presence of LVH is an indication to initiate or intensify antihypertensive therapy.*

Hypertension is associated with increased risk of myocardial infarction, stroke, and cardiovascular mortality in adults,<sup>2,51</sup> and treatment of elevated BP results in a reduction in the risk for cardiovascular events.

Children and adolescents with severe elevation of BP are also at increased risk of adverse outcomes, including hypertensive encephalopathy, seizures, and even cerebrovascular accidents and congestive heart failure.<sup>52,53</sup> Even hypertension that is less severe contributes to target-organ damage when it occurs with other chronic conditions, such as chronic kidney disease.<sup>54–56</sup> Two autopsy studies,<sup>57,58</sup> that evaluated tissue from adolescents and young adults who had sudden deaths due to trauma, demonstrated significant relationships between the level of BP, or hypertension, and the presence of atherosclerotic lesions in the aorta and coronary arteries. The exact level and duration of BP elevation that causes target-organ damage in the young has not been established.

One difficulty in the assessment of these relationships is that, until recently, few non-invasive methods could evaluate the effect of hypertension on the cardiovascular system. Noninvasive techniques that use ultrasound can demonstrate structural and functional changes in the vasculature related to BP.

Recent clinical studies using these techniques demonstrate that childhood levels of BP are associated with carotid intimal-medial thickness<sup>59</sup> and large artery compliance<sup>60</sup> in young adults. Even healthy adolescents with clustering of cardiovascular risk factors demonstrate elevated carotid thickness,<sup>61,62</sup> and those with BP levels at the higher end of the normal distribution show decreased brachial artery flow-mediated vasodilatation. Overall, evidence is increasing that even mild BP elevation can have an adverse effect on vascular structure and function<sup>63</sup> in asymptomatic young persons.

LVH is the most prominent clinical evidence of target-organ damage caused by hypertension in children and adolescents. With the use of echocardiography to measure left ventricular mass, LVH has been reported in 34–38 percent of children and adolescents with mild, untreated BP elevation.<sup>64–66</sup> Daniels et al. evaluated 130 children and adolescents with persistent BP elevation.<sup>67</sup> They reported that 55 percent of patients had a left ventricular mass index greater than the 90th percentile, and 14 percent had left ventricular mass index above 51 g/m<sup>2.7</sup>, a value in adults with hypertension that has been associated with a fourfold greater risk of adverse cardiovascular outcomes. When left ventricular geometry was examined in hypertensive children,

17 percent had concentric hypertrophy, a pattern that is associated with higher risk for cardiovascular outcomes in adults, and 30 percent had eccentric hypertrophy, which is associated with intermediate risk for cardiovascular outcomes.<sup>67</sup>

In addition, abnormalities of the retinal vasculature have been reported in adults with hypertension.<sup>68</sup> Few studies of retinal abnormalities have been conducted in children with hypertension. Skalina et al. evaluated newborns with hypertension<sup>69</sup> and reported the presence of hypertensive retinal abnormalities in approximately 50 percent of their patients. On repeat examination, after the resolution of hypertension, these abnormalities had disappeared.

## CLINICAL RECOMMENDATION

Echocardiography is recommended as a primary tool for evaluating patients for target-organ abnormalities by assessing the presence or absence of LVH. Left ventricular mass is determined from standard echocardiographic measurements of the left ventricular end-diastolic dimension (LVED), the intraventricular septal thickness (IVS), and the thickness of the left ventricular posterior wall (LVPW) and can be calculated as:  $LV\ Mass\ (g) = 0.80 [1.04 (IVS + LVED + LVPW)^3 - (LVED)^3] + 0.6$  (with echocardiographic measurements in centimeters). From these measures, the left ventricular mass can be calculated by using the equation of Devereux et al.<sup>70</sup> when measurements are made according to the criteria of the American Society of Echocardiography.<sup>71</sup>

Heart size is closely associated with body size.<sup>72</sup> Left ventricular mass index is calculated to standardize measurements of left ventricular mass. Several methods for indexing left ventricular mass have been reported, but it is recommended that height

( $m^{2.7}$ ) be used to index left ventricular mass, as described by de Simone et al.<sup>73</sup> This method accounts for close to the equivalent of the effect of lean body mass and excludes the effect of obesity and BP elevation on left ventricular mass. Some echo laboratories use height as the indexing variable. This calculation is also acceptable and is somewhat easier to use, as fewer calculations are needed.

Children and adolescents with established hypertension should have an echocardiogram to determine if LVH is present. A conservative cutpoint that determines the presence of LVH is  $51\ g/m^{2.7}$ . This cutpoint is above the 99th percentile for children and adolescents and is associated with increased morbidity in adults with hypertension.<sup>73</sup> Other references exist for normal children,<sup>74</sup> but, unlike adults, outcome-based standards for left ventricular mass index are not available for children. In interpreting the left ventricular mass index, it should be remembered that some factors, such as obesity and hypertension, have pathologic effects on the heart, whereas others—such as physical activity, particularly in highly conditioned athletes—may be adaptive.

Ascertainment of left ventricular mass index is very helpful in clinical decisionmaking. The presence of LVH can be an indication for initiating or intensifying pharmacologic therapy to lower BP. For patients who have LVH, the echocardiographic determination of left ventricular mass index should be repeated periodically.

At the present time, additional testing for other target-organ abnormalities (such as determination of carotid intimal-medial thickness and evaluation of urine for microalbuminuria) is not recommended for routine clinical use. Further research will be needed to evaluate the clinical utility of these tests.

# Therapeutic Lifestyle Changes

- *Weight reduction is the primary therapy for obesity-related hypertension. Prevention of excess or abnormal weight gain will limit future increases in BP.*
- *Regular physical activity and restriction of sedentary activity will improve efforts at weight management and may prevent an excess increase in BP over time.*
- *Dietary modification should be strongly encouraged in children and adolescents who have BP levels in the prehypertensive range as well as in those with hypertension.*
- *Family-based intervention improves success.*

Evidence is limited that supports the efficacy of nonpharmacological interventions for BP reduction in the treatment of hypertension in children and adolescents. Data that demonstrate a relationship of lifestyle with BP can be used as the basis for recommendations. On the basis of large randomized controlled trials, the following lifestyle modifications are recommended in adults:<sup>2</sup> weight reduction in overweight or obese individuals;<sup>75</sup> increased intake of fresh vegetables, fruits, and lowfat dairy (the Dietary Approaches to Stop Hypertension Study [DASH] eating plan);<sup>76</sup> dietary sodium reduction;<sup>76,77</sup> increased physical activity;<sup>78</sup> and moderation of alcohol consumption.<sup>79</sup> Smoking cessation has significant cardiovascular benefits.<sup>32</sup> As information on chronic sleep problems evolves, interventions to improve sleep quality may also have a beneficial effect on BP.<sup>80</sup>

The potential for control of BP in children through weight reduction is supported by BP tracking and weight-reduction studies. BP levels track from childhood through adolescence and into adulthood<sup>81–83</sup> in association with weight.<sup>84,85</sup> Because of the strong correlation between weight and BP, excessive weight gain is likely to be associated with elevated BP over time. Therefore, maintenance of normal weight gain in childhood should lead to less hypertension in adulthood.

Weight loss in overweight adolescents is associated with a decrease in BP.<sup>30,86–90</sup> Weight control not only decreases BP, it also decreases BP sensitivity to salt<sup>88</sup> and decreases other cardiovascular risk factors, such as dyslipidemia and insulin resistance.<sup>32</sup> In studies that achieve a reduction in BMI of about 10 percent, short-term reductions in BP were in the range of 8–12 mmHg. Although difficult, weight loss, if successful, is extremely effective.<sup>32,91–93</sup> Identifying a complication of overweight, such as hypertension, can be a helpful motivator for patients and families to make changes. Weight control can render pharmacological treatment unnecessary but should not delay drug use when indicated.

Emphasis on the management of complications rather than on overweight shifts the aim of weight management from an aesthetic to a health goal. In motivated families, education or simple behavior modification can be successful in achieving moderate weight loss or preventing further weight gain. Steps can be implemented in the primary care setting even with limited staff and time resources.<sup>32,91</sup> The patient should be encouraged to self-monitor time spent in sedentary activity, including watching television and playing video or computer games, and to set goals to progressively decrease these activities to less than 2 hours per day.<sup>94</sup> The family and patient should identify physical activities that the child enjoys,

engage in them regularly, and self-monitor time spent in physical activities (30–60 minutes per day should be achieved).<sup>94–96</sup> Dietary changes can involve portion-size control, decrease in consumption of sugar-containing beverages and energy-dense snacks, increase in consumption of fresh fruits and vegetables, and regular meals including a healthy breakfast.<sup>32,91,93,97,98</sup> Consultation with a nutritionist can be useful and provide customized recommendations. During regular office visits, the primary care provider can supervise the child's progress in self-monitoring and accomplishing goals and can provide support and positive feedback to the family. Some patients will benefit from a more intense and comprehensive approach to weight management from a multidisciplinary and specialized team if available.<sup>91–93</sup>

Despite the lack of firm evidence about dietary intervention in children, it is generally accepted that hypertensive individuals can benefit from a dietary increase in fresh vegetables, fresh fruits, fiber, and nonfat dairy, as well as a reduction of sodium. Despite some suggestion that calcium supplements may decrease BP in children,<sup>99,100</sup> so far the evidence is too limited to support a clinical recommendation.<sup>101</sup> Lower BP has been associated in children and adolescents with an *increased* intake of potassium,<sup>100–103</sup> magnesium,<sup>100,101</sup> folic acid,<sup>101,104</sup> unsaturated fat,<sup>100,105,106</sup> and fiber,<sup>100,101,104</sup> and lower dietary intake of total fat.<sup>100,101</sup> However, these associations are small and insufficient to support dietary recommendations for specific, individual nutrients.

Sodium reduction in children and adolescents has been associated with small reductions in BP, in the range of 1–3 mmHg.<sup>101,103,107–110</sup> Data from one randomized trial suggest that sodium intake in infancy may affect BP in adolescence.<sup>111</sup> Similarly, some evidence indicates that breastfeeding may be associated with lower BP in childhood.<sup>112,113</sup> The current recommendation for adequate daily sodium intake is only 1.2 g/day for 4- to 8-year-old children and 1.5 g/day for older children.<sup>114</sup>

Since this amount of sodium is substantially lower than current dietary intakes, lowering dietary sodium from the current usual intake may have future benefit. Reduced sodium intake, with calorie restriction, may account for some of the BP improvement associated with weight loss.

Regular physical activity has cardiovascular benefits. A recent meta-analysis that combined 12 randomized trials, for a total of 1,266 children and adolescents, concluded that physical activity leads to a small, but not statistically significant, decrease in BP.<sup>115</sup> However, both regular physical activity and decreasing sedentary activity—such as watching television and playing video or electronic games—are important components of pediatric obesity treatment and prevention.<sup>32,91–93</sup> Weight-reduction trials consistently report better results when physical activity and/or prevention of sedentary activity are included in the treatment protocol. Therefore, regular aerobic physical activity (30–60 minutes of moderate physical activity on most days) and limitation of sedentary activities to less than 2 hours per day are recommended for the prevention of obesity, hypertension, and other cardiovascular risk factors.<sup>94–96</sup> With the exception of power lifting, resistance training is also helpful. Competitive sports participation should be limited only in the presence of uncontrolled Stage 2 hypertension.<sup>116</sup>

The scope of hypertension as a public health problem in adults is substantial. Poor health-related behaviors such as physical inactivity, unfavorable dietary patterns, and excessive weight gain raise the risk for future hypertension. The therapeutic lifestyle changes discussed above may have benefit for all children in prevention of future disease, including primary hypertension. Accordingly, appropriate health recommendations for all children and adolescents are regular physical activity; a diet with limited sodium but rich in fresh fruits, fresh vegetables, fiber, and lowfat dairy; and avoiding excess weight gain.

# Pharmacologic Therapy of Childhood Hypertension

- *Indications for antihypertensive drug therapy in children include secondary hypertension and insufficient response to lifestyle modifications.*
- *Recent clinical trials have expanded the number of drugs that have pediatric dosing information. Dosing recommendations for many of the newer drugs are provided.*
- *Pharmacologic therapy, when indicated, should be initiated with a single drug. Acceptable drug classes for use in children include ACE inhibitors, angiotensin-receptor blockers, beta-blockers, calcium channel blockers, and diuretics.*
- *The goal for antihypertensive treatment in children should be reduction of BP to <95th percentile, unless concurrent conditions are present. In that case, BP should be lowered to <90th percentile.*
- *Severe, symptomatic hypertension should be treated with intravenous antihypertensive drugs.*

In adults, hypertension is typically a lifelong condition. Most hypertensive patients will need to remain on medications for the rest of their lives. Usually, adults readily accept this fact, given the known long-term adverse consequences of untreated or undertreated hypertension.<sup>117</sup> In children, however, the long-term consequences of untreated hypertension are unknown. Additionally, no data are available on the long-term effects of antihypertensive drugs on growth and development. Therefore, a definite indication for initiating pharmacologic therapy should be ascertained before a drug is prescribed.

Table 6 summarizes the indications for use of antihypertensive drugs in children. These indications include symptomatic hypertension, secondary hypertension, established hypertensive target-organ damage, and failure of nonpharmacologic measures. Other indications for use of antihypertensive drugs can be considered, depending on the clinical situation. For example, because the presence of multiple cardiovascular risk factors (elevated BP, dyslipidemia, tobacco use, etc.) increases cardiovascular risk in an exponential rather

than additive fashion,<sup>118,119</sup> antihypertensive therapy could be considered if the child or adolescent is known to have dyslipidemia.

The number of antihypertensive drugs has increased since the publication of the *Report of the Task Force on Blood Pressure Control in Children*.<sup>120</sup> The number of drugs that have been studied systematically in children has also increased, largely because of incentives provided to the pharmaceutical industry under the auspices of the 1997 Food and Drug Administration Modernization Act (FDAMA) and the 2002 Best Pharmaceuticals for Children Act (BPCA).<sup>121–123</sup> These developments have had both negative and positive consequences. Chief among the negative consequences is the lack of reliable pediatric data for older, commonly used compounds with expired patent protection. Currently, no incentives exist for industry-sponsored trials of such drugs, and alternative methods of stimulating pediatric studies, such as those contained in the BPCA,<sup>123–125</sup> have yet to come to fruition. On the other hand, publication of the results of industry-sponsored clinical trials and single-center case series will

provide additional data that can be combined with prior recommendations based on expert opinion and collective clinical experience to guide the use of antihypertensive drugs in children and adolescents who require pharmacologic treatment.

Table 9 contains dosing recommendations for antihypertensive drugs in children 1–17 years old. It should be noted that many other drugs are available in addition to those listed in table 9. Those drugs are not included in the table, however, because few or no pediatric data were available at the time this report was prepared.

Long-term, clinical endpoint data from randomized trials, such as the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT), support the preferential use of specific antihypertensive drugs in adults.<sup>2,126</sup> However, pediatric clinical trials of antihypertensive drugs have focused only on their ability to lower BP and have not compared the effects of these drugs on clinical endpoints. Therefore, because all classes of antihypertensive drugs have been shown to lower BP in children, the choice of drug for initial antihypertensive therapy resides in the preference of the responsible physician. Some diuretics and beta-adrenergic blockers, which were recommended as initial therapy in the first and second Task Force Reports,<sup>25,120</sup> have a long history of safety and efficacy based on clinical experience in hypertensive children, and these drugs remain appropriate for pediatric use. Similarly, some members of the newer classes of antihypertensive drugs, including ACE inhibitors, calcium channel blockers, and angiotensin-receptor blockers,<sup>127–130</sup> have been studied in children and, based on short-term use, shown to be safe and well-tolerated with satisfactory BP reductions in hypertensive children.

Specific classes of antihypertensive drugs should be used preferentially in certain hypertensive children with specific underlying or concurrent medical conditions. Examples include the use of ACE inhibitors or angiotensin-receptor blockers in children with diabetes and microalbuminuria or proteinuric renal diseases, and the use of beta-adrenergic

blockers or calcium channel blockers in hypertensive children with migraine headaches. This approach is similar to that outlined in the recent JNC 7 report, which recommends specific classes of antihypertensive drugs for use in adults in certain high-risk categories.<sup>2</sup>

All antihypertensive drugs should be prescribed in a similar fashion: The child is initially started on the lowest recommended dose listed in table 9. The dose can be increased until the desired BP goal is achieved. Once the highest recommended dose is reached, or if the child experiences side effects from the drug, a second drug from a different class should be added. Consideration should be given to combining drugs with complementary mechanisms of action, such as an ACE inhibitor with a diuretic, or a vasodilator with a diuretic or beta-adrenergic blocker. Because little pediatric experience is available in using fixed-dose combination products, except for bisoprolol/HCTZ,<sup>131</sup> routine use of these products in children cannot be recommended at this time.

For children with uncomplicated primary hypertension and no hypertensive target-organ damage, the goal BP should be less than the 95th percentile for sex, age, and height, whereas for children with chronic renal disease, diabetes, or hypertensive target-organ damage, the goal BP should be less than the 90th percentile for sex, age, and height. Again, this approach is similar to the recommended treatment of hypertension in adults with additional cardiovascular risk factors or comorbid conditions.<sup>2</sup>

Important adjunctive aspects to the drug therapy of childhood hypertension include ongoing monitoring of target-organ damage as well as BP monitoring, surveillance for drug side effects, periodic monitoring of electrolytes in children treated with ACE inhibitors or diuretics, counseling regarding other cardiovascular risk factors, and continued emphasis on nonpharmacologic measures. It may also be appropriate to consider “step-down” therapy in selected patients. This approach attempts a gradual reduction in the drug after an extended course of good BP control, with

TABLE 9

## Antihypertensive Drugs for Outpatient Management of Hypertension in Children 1–17 Years Old\*

Class	Drug	Dose†
Angiotensin-converting enzyme (ACE) inhibitor	Benazepril	Initial: 0.2 mg/kg/day up to 10 mg/day Maximum: 0.6 mg/kg/day up to 40 mg/day
	Captopril	Initial: 0.3–0.5 mg/kg/dose Maximum: 6 mg/kg/day
	Enalapril	Initial: 0.08 mg/kg/day up to 5 mg/day Maximum: 0.6 mg/kg/day up to 40 mg/day
	Fosinopril	Children >50 kg: Initial: 5–10 mg/day Maximum: 40 mg/day
	Lisinopril	Initial: 0.07 mg/kg/day up to 5 mg/day Maximum: 0.6 mg/kg/day up to 40 mg/day
	Quinapril	Initial: 5–10 mg/day Maximum: 80 mg/day
Angiotensin-receptor blocker	Irbesartan	6–12 years: 75–150 mg/day ≥13 years: 150–300 mg/day
	Losartan	Initial: 0.7 mg/kg/day up to 50 mg/day Maximum: 1.4 mg/kg/day up to 100 mg/day
α- and β-blocker	Labetalol	Initial: 1–3 mg/kg/day Maximum: 10–12 mg/kg/day up to 1,200 mg/day
β-blocker	Atenolol	Initial: 0.5–1 mg/kg/day Maximum: 2 mg/kg/day up to 100 mg/day
	Bisoprolol/HCTZ	Initial: 2.5/6.25 mg/day Maximum: 10/6.25 mg/day
	Metoprolol	Initial: 1–2 mg/kg/day Maximum: 6 mg/kg/day up to 200 mg/day
	Propranolol	Initial: 1–2 mg/kg/day Maximum: 4 mg/kg/day up to 640 mg/day
Calcium channel blocker	Amlodipine	Children 6–17 years: 2.5–5 mg once daily
	Felodipine	Initial: 2.5 mg/day Maximum: 10 mg/day
	Isradipine	Initial: 0.15–0.2 mg/kg/day Maximum: 0.8 mg/kg/day up to 20 mg/day
	Extended-release nifedipine	Initial: 0.25–0.5 mg/kg/day Maximum: 3 mg/kg/day up to 120 mg/day

Dosing Interval	Evidence‡	FDA Labelings§	Comments††
qd	RCT	Yes	1. All ACE inhibitors are contraindicated in pregnancy—females of childbearing age should use reliable contraception.
tid	RCT, CS	No	2. Check serum potassium and creatinine periodically to monitor for hyperkalemia and azotemia.
qd–bid	RCT	Yes	3. Cough and angioedema are reportedly less common with newer members of this class than with captopril.
qd	RCT	Yes	4. Benazepril, enalapril, and lisinopril labels contain information on the preparation of a suspension; captopril may also be compounded into a suspension.
qd	RCT	Yes	5. FDA approval for ACE inhibitors with pediatric labeling is limited to children ≥6 years of age and to children with creatinine clearance ≥30 ml/min/1.73m <sup>2</sup> .
qd	RCT, EO	No	
qd	CS	Yes	1. All ARBs are contraindicated in pregnancy—females of childbearing age should use reliable contraception.
qd	RCT	Yes	2. Check serum potassium, creatinine periodically to monitor for hyperkalemia and azotemia. 3. Losartan label contains information on the preparation of a suspension. 4. FDA approval for ARBs is limited to children ≥6 years of age and to children with creatinine clearance ≥30 ml/min/1.73m <sup>2</sup> .
bid	CS, EO	No	1. Asthma and overt heart failure are contraindications. 2. Heart rate is dose-limiting. 3. May impair athletic performance. 4. Should not be used in insulin-dependent diabetics.
qd–bid	CS	No	1. Noncardioselective agents (propranolol) are contraindicated in asthma and heart failure.
qd	RCT	No	2. Heart rate is dose-limiting. 3. May impair athletic performance.
bid	CS	No	4. Should not be used in insulin-dependent diabetics.
bid–tid	RCT, EO	Yes	5. A sustained-release formulation of propranolol is available that is dosed once-daily.
qd	RCT	Yes	1. Amlodipine and isradipine can be compounded into stable extemporaneous suspensions.
qd	RCT, EO	No	2. Felodipine and extended-release nifedipine tablets must be swallowed whole.
tid–qid	CS, EO	No	3. Isradipine is available in both immediate-release and sustained-release formulations; sustained release form is dosed qd or bid.
qd–bid	CS, EO	No	4. May cause tachycardia.

TABLE 9

**Antihypertensive Drugs for Outpatient Management of Hypertension in Children 1–17 Years Old\*  
(continued)**

Class	Drug	Dose†
Central $\alpha$ -agonist	Clonidine	Children $\geq 12$ years: Initial: 0.2 mg/day Maximum: 2.4 mg/day
Diuretic	HCTZ	Initial: 1 mg/kg/day Maximum: 3 mg/kg/day up to 50 mg/day
	Chlorthalidone	Initial: 0.3 mg/kg/day Maximum: 2 mg/kg/day up to 50 mg/day
	Furosemide	Initial: 0.5–2.0 mg/kg/dose Maximum: 6 mg/kg/day
	Spironolactone	Initial: 1 mg/kg/day Maximum: 3.3 mg/kg/day up to 100 mg/day
	Triamterene	Initial: 1–2 mg/kg/day Maximum: 3–4 mg/kg/day up to 300 mg/day
	Amiloride	Initial: 0.4–0.625 mg/kg/day Maximum: 20 mg/day
Peripheral $\alpha$ -antagonist	Doxazosin	Initial: 1 mg/day Maximum: 4 mg/day
	Prazosin	Initial: 0.05–0.1 mg/kg/day Maximum: 0.5 mg/kg/day
	Terazosin	Initial: 1 mg/day Maximum: 20 mg/day
Vasodilator	Hydralazine	Initial: 0.75 mg/kg/day Maximum: 7.5 mg/kg/day up to 200 mg/day
	Minoxidil	Children $< 12$ years: Initial: 0.2 mg/kg/day Maximum: 50 mg/day Children $\geq 12$ years: Initial: 5 mg/day Maximum: 100 mg/day

Dosing Interval	Evidence‡	FDA Labelings§	Comments††
bid	EO	Yes	<ol style="list-style-type: none"> <li>1. May cause dry mouth and/or sedation.</li> <li>2. Transdermal preparation also available.</li> <li>3. Sudden cessation of therapy can lead to severe rebound hypertension.</li> </ol>
qd	EO	Yes	<ol style="list-style-type: none"> <li>1. All patients treated with diuretics should have electrolytes monitored shortly after initiating therapy and periodically thereafter.</li> </ol>
qd	EO	No	<ol style="list-style-type: none"> <li>2. Useful as add-on therapy in patients being treated with drugs from other drug classes.</li> </ol>
qd–bid	EO	No	<ol style="list-style-type: none"> <li>3. Potassium-sparing diuretics (spironolactone, triamterene, amiloride) may cause severe hyperkalemia, especially if given with ACE inhibitor or ARB.</li> </ol>
qd–bid	EO	No	<ol style="list-style-type: none"> <li>4. Furosemide is labeled only for treatment of edema but may be useful as add-on therapy in children with resistant hypertension, particularly in children with renal disease.</li> </ol>
bid	EO	No	<ol style="list-style-type: none"> <li>5. Chlorthalidone may precipitate azotemia in patients with renal diseases and should be used with caution in those with severe renal impairment.</li> </ol>
qd	EO	No	
qd	EO	No	<ol style="list-style-type: none"> <li>1. May cause hypotension and syncope, especially after first dose.</li> </ol>
tid	EO	No	
qd	EO	No	
qid	EO	Yes	<ol style="list-style-type: none"> <li>1. Tachycardia and fluid retention are common side effects.</li> </ol>
qd–tid	CS, EO	Yes	<ol style="list-style-type: none"> <li>2. Hydralazine can cause a lupus-like syndrome in slow acetylators.</li> <li>3. Prolonged use of minoxidil can cause hypertrichosis.</li> <li>4. Minoxidil is usually reserved for patients with hypertension resistant to multiple drugs.</li> </ol>

ACE, angiotensin-converting enzyme; ARB, angiotensin-receptor blocker; bid, twice-daily; HCTZ, hydrochlorothiazide; qd, once-daily; qid, four times daily; tid, three times daily

\* Includes drugs with prior pediatric experience or recently completed clinical trials.

† The maximum recommended adult dose should not be exceeded in routine clinical practice.

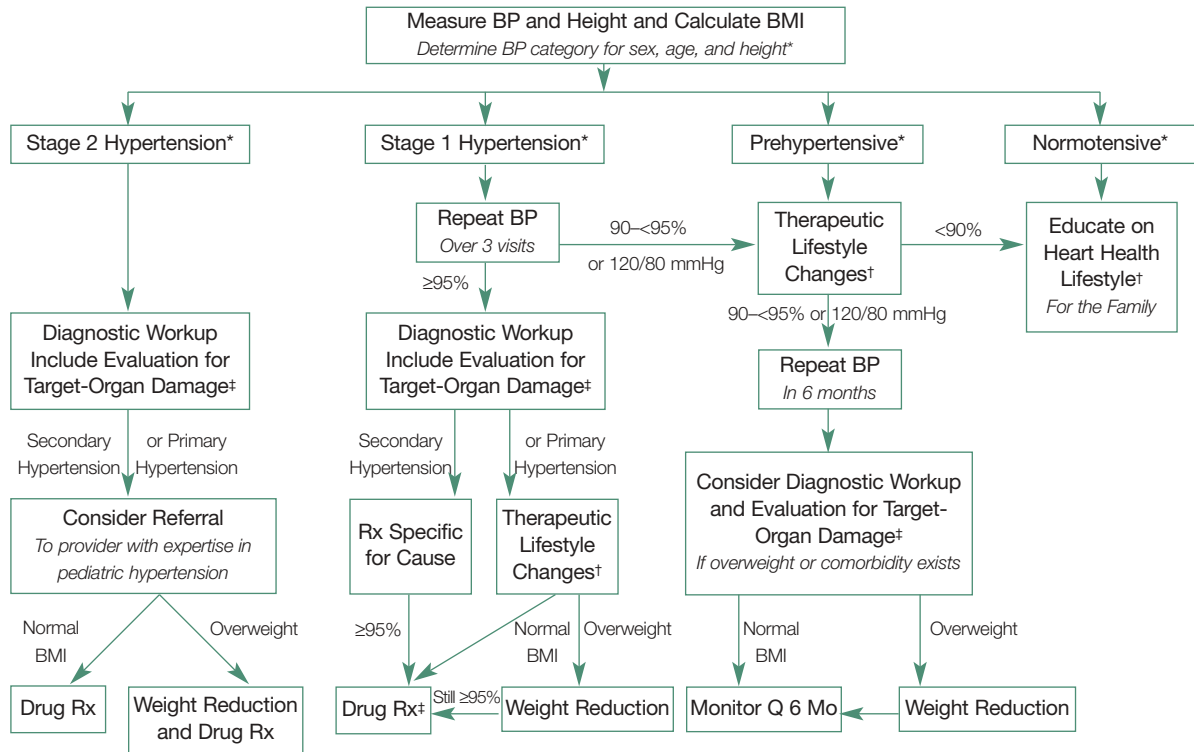
‡ Level of evidence upon which dosing recommendations are based (CS, case series; EO, expert opinion; RCT, randomized controlled trial)

§ FDA-approved pediatric labeling information is available. Recommended doses for agents with FDA-approved pediatric labels are the doses contained in the approved labels. Even when pediatric labeling information is not available, the FDA-approved label should be consulted for additional safety information.

†† Comments apply to all members of each drug class except where otherwise stated.

FIGURE 1

Management Algorithm



BMI, body mass index; BP, blood pressure; Rx, prescription; Q, every.

\*See tables 3, 4, and 5.

†Diet modification and physical activity.

‡Especially if younger, very high BP, little or no family history, diabetic, or other risk factors.

the eventual goal of completely discontinuing drug therapy. Children with uncomplicated primary hypertension, especially overweight children who successfully lose weight, are the best candidates for the step-down approach. Such patients require ongoing BP monitoring after the cessation of drug therapy, as well as continued nonpharmacologic treatment, because hypertension may recur.

Severe, symptomatic hypertension with BP well above the 99th percentile occurs in some children, usually those with underlying renal disease, and requires prompt treatment. *Hypertensive emergencies* in children are usually accompanied by signs of hypertensive encephalopathy, typically causing seizures. Hypertensive emergencies should be treated by an intravenous antihypertensive that can produce a controlled reduction in BP, aiming to decrease the pressure by 25 percent or less over the first 8 hours after presentation and then gradually normalizing the BP over 26–48

hours.<sup>132,133</sup> *Hypertensive urgencies* are accompanied by less serious symptoms, such as severe headache or vomiting. Hypertensive urgencies can be treated by either intravenous or oral antihypertensives, depending on the child’s symptomatology. Table 10 provides dosing recommendations for treatment of severe hypertension in children when prompt reduction in BP is indicated.

Figure 1 is a management algorithm that presents guidelines for evaluation and treatment of Stage 1 and Stage 2 hypertension in children and adolescents. The algorithm summarizes monitoring and intervention recommendations for children and adolescents with prehypertension and hypertension. Included in the algorithm are points at which the presence of overweight is considered in clinical decisionmaking. The algorithm also emphasizes the inclusion of evaluation for target-organ damage in children with established Stage 1 and Stage 2 hypertension.

TABLE 10

### Antihypertensive Drugs for Management of Severe Hypertension in Children 1–17 Years Old

Most Useful*				
Drug	Class	Dose†	Route	Comments
Esmolol	β-blocker	100–500 mcg/kg/min	iv infusion	Very short-acting—constant infusion preferred. May cause profound bradycardia. Produced modest reductions in BP in a pediatric clinical trial.
Hydralazine	Vasodilator	0.2–0.6 mg/kg/dose	iv, im	Should be given every 4 hours when given iv bolus. Recommended dose is lower than FDA label.
Labetalol	α- and β-blocker	bolus: 0.2–1.0 mg/kg/dose up to 40 mg/dose infusion: 0.25–3.0 mg/kg/hr	iv bolus or infusion	Asthma and overt heart failure are relative contraindications.
Nicardipine	Calcium channel blocker	1–3 mcg/kg/min	iv infusion	May cause reflex tachycardia.
Sodium nitroprusside	Vasodilator	0.53–10 mcg/kg/min	iv infusion	Monitor cyanide levels with prolonged (>72 hr) use or in renal failure; or coadminister with sodium thiosulfate.
Occasionally Useful‡				
Drug	Class	Dose*	Route	Comments
Clonidine	Central α-agonist	0.05–0.1 mg/dose may be repeated up to 0.8 mg total dose	po	Side effects include dry mouth and sedation.
Enalaprilat	ACE inhibitor	0.05–0.1 mg/kg/dose up to 1.25 mg/dose	iv bolus	May cause prolonged hypotension and acute renal failure, especially in neonates.
Fenoldopam	Dopamine receptor agonist	0.2–0.8 mcg/kg/min	iv infusion	Produced modest reductions in BP in a pediatric clinical trial in patients up to 12 years.
Isradipine	Calcium channel blocker	0.05–0.1 mg/kg/dose	po	Stable suspension can be compounded.
Minoxidil	Vasodilator	0.1–0.2 mg/kg/dose	po	Most potent oral vasodilator; long-acting.

ACE, angiotensin-converting enzyme; im, intramuscular; iv, intravenous; po, oral.

\* Useful for hypertensive emergencies and some hypertensive urgencies.

† All dosing recommendations are based upon expert opinion or case series data except as otherwise noted.

‡ Useful for hypertensive urgencies and some hypertensive emergencies.

**APPENDIX A.**  
**Demographic Data**

**TABLE A-1**

**Demographic Data on Height/Blood Pressure Distribution Curves by Study Population\***

Age Source	(Years)	Gender		Ethnic Group					Missing	Person's Visits SBP Available	Person's Visits DBP.5 Available	Total No. of Person's Visits	
		Boys	Girls	Black	Hispanic	White	Asian	Native American					Other
NIH	6-17	1,896	1,751	600	0	2,963	0	0	84	0	3,647	3,609	3,647
Pittsburgh	1-5	148	137	108	0	176	0	0	0	1	285	0	285
Dallas	13-17	5,916	5,649	5,266	1,570	4,729	0	0	0	0	11,565	11,565	11,565
Bogalusa	1-17	3,751	3,607	2,480	0	4,878	0	0	0	0	7,358	0	7,358
Houston	3-17	1,457	1,377	637	1,341	748	23	0	0	85	2,834	0	2,834
South Carolina	4-17	3,167	3,263	3,110	0	3,320	0	0	0	0	6,430	6,368	6,430
Iowa	5-17	2,099	1,993	0	0	4,092	0	0	0	0	4,092	0	4,092
Providence	1-3	230	231	24	4	431	0	0	2	0	461	371	461
Minnesota	9-17	9,991	9,418	3,422	555	11,311	1,677	644	1,800	0	19,409	19,207	19,409
NHANES III	5-17	2,465	2,577	1,770	1,830	1,324	64	10	12	32	5,042	4,304	5,042
NHANES 99-00	8-17	1,041	1,063	605	988	437	0	0	74	0	2,104	2,076	2,104
<b>Total (Percent of Total)</b>	<b>1-17</b>	<b>32,161 (51)</b>	<b>31,066 (49)</b>	<b>18,022 (29)</b>	<b>6,288 (10)</b>	<b>34,409 (54)</b>	<b>1,764 (3)</b>	<b>654 (1)</b>	<b>1,972 (3)</b>	<b>118 (0)</b>	<b>63,227 (83,091)</b>	<b>47,500 (57,976)</b>	<b>63,227 (83,091)</b>

DBP.5, diastolic blood pressure (Korotkoff 5); NHANES, National Health and Nutrition Examination Survey; NIH, National Institutes of Health; SBP, systolic blood pressure.

\* Table differs from 1997 report: updated height percentile used; subjects whose height Z-score was less than -6 or greater than +6 were excluded.

**APPENDIX B.**

**Computation of Blood Pressure  
Percentiles for Arbitrary Sex, Age,  
and Height**

# Computation of Blood Pressure Percentiles for Arbitrary Sex, Age, and Height

- To compute the systolic blood pressure (SBP) percentile of a boy who is age  $y$  years and height  $h$  inches with SBP =  $x$  mmHg:

1. Refer to the most recent CDC growth charts, which are available online, and convert the height of  $h$  inches to a height Z-score relative to boys of the same age; this is denoted by  $Zht$ .

2. Compute the expected SBP ( $\mu$ ) for boys of age  $y$  years and height  $h$  inches given by

$$\mu = \alpha + \sum_{j=1}^4 \beta_j (y-10)^j + \sum_{k=1}^4 \gamma_k (Zht)^k$$

where  $\alpha, \beta_1, \dots, \beta_4$  and  $\gamma_1, \dots, \gamma_4$  are given in the 3rd column of appendix table B-1.

3. Then convert the boy's observed SBP to a Z-score ( $Zbp$ ) given by

$$Zbp = (x - \mu) / \sigma$$

where  $\sigma$  is given in the 3rd column of appendix table B-1.

4. To convert the bp Z-score to a percentile (P), compute  $P = \Phi(Zbp) \times 100\%$  where  $\Phi(Z)$  = area under a standard normal distribution to the left of Z.

Thus, if  $Zbp = 1.28$ , then  $\Phi(Zbp) = .90$  and the bp percentile =  $.90 \times 100\% = 90\%$ .

5. To compute percentiles for SBP for girls, diastolic blood pressure (DBP) (K5) for boys, and DBP (K5) for girls, use the regression coefficients from the 4th, 5th, and 6th columns of appendix table B-1.

For example, a 12-year-old boy, with height at the 90th percentile for his age-sex group, has a height Z-score = 1.28, and his expected SBP ( $\mu$ ) is

$$\mu = 102.19768 + 1.82416 (2) + 0.12776 (2^2) + 0.00249 (2^3) - 0.00135 (2^4) + 2.73157 (1.28) - 0.19618 (1.28)^2 - 0.04659 (1.28)^3 + 0.00947 (1.28)^4 = 109.46 \text{ mmHg.}$$

Suppose his actual SBP is 120 mmHg ( $x$ ); his SBP Z-score is then:

$$\text{SBP Z-score} = (x - \mu) / \sigma = (120 - 109.46) / 10.7128 = 0.984$$

The corresponding SBP percentile =  $\Phi(0.984) \times 100\% = 83.7\text{th}$  percentile.

TABLE B-1

## Regression Coefficients From Blood Pressure Regression Models\*

Variable Name	Symbol	Systolic BP		Diastolic BP5	
		Male	Female	Male	Female
Intercept	$\alpha$	102.19768	102.01027	61.01217	60.50510
Age					
Age-10	$\beta_1$	1.82416	1.94397	0.68314	1.01301
(Age-10) <sup>2</sup>	$\beta_2$	0.12776	0.00598	-0.09835	0.01157
(Age-10) <sup>3</sup>	$\beta_3$	0.00249	-0.00789	0.01711	0.00424
(Age-10) <sup>4</sup>	$\beta_4$	-0.00135	-0.00059	0.00045	-0.00137
Normalized height					
Zht	$\gamma^1$	2.73157	2.03526	1.46993	1.16641
Zht <sup>2</sup>	$\gamma^2$	-0.19618	0.02534	-0.07849	0.12795
Zht <sup>3</sup>	$\gamma^3$	-0.04659	-0.01884	-0.03144	-0.03869
Zht <sup>4</sup>	$\gamma^4$	0.00947	0.00121	0.00967	-0.00079
Standard deviation	$\sigma$	10.7128	10.4855	11.6032	10.9573
$\rho^\dagger$		0.4100	0.3824	0.2436	0.2598
n (persons)		32,161	31,066	24,057	23,443
n (visits)		42,074	41,017	29,182	28,794

BP, blood pressure; Diastolic BP5, diastolic measurement at Korotkoff 5.

\* The coefficients were obtained from mixed-effects linear regression models.

† The value of  $\rho$  represents the correlation between BP measurements at different ages for the same child after correcting for age and Zht. This computation was necessary because some studies contributing to the childhood BP database provided BP at more than one age.

# Scheme Used for Classification of the Evidence

- M** Meta-analysis; use of statistical methods to combine the results from clinical trials
- RA** Randomized controlled trials; also known as experimental studies
- RE** Retrospective analyses; also known as case-control studies
- F** Prospective study; also known as cohort studies, including historical or prospective followup studies
- X** Cross-sectional survey; also known as prevalence studies
- PR** Previous review or position statements
- C** Clinical interventions (nonrandomized)

These symbols are appended to the citations in the reference list. The studies that provided evidence supporting the recommendations of this report were classified and reviewed by the staff and the executive committee. The classification scheme is from the JNC 7 report and other NHBPEP Working Group Reports ([www.nhlbi.nih.gov/about/nhbpep/index.htm](http://www.nhlbi.nih.gov/about/nhbpep/index.htm)).<sup>2,134-138</sup>

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## Commentary

## Portion Size Me: Downsizing Our Consumption Norms

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We are a nation of super-sized portions and super-sized people. When faced with the family-sized box of breakfast cereal, five sizes of french fries, or a 14-in dinner plate, it is easy to forget our world history. In 75 years we have gone from shivering in Depression breadlines and hoarding food-ration stamps to being an overweight, super-sized country. Most of us are now surrounded with a portion-distorted embarrassment of food.

We find portion distortions in supermarkets, where the number of larger sizes has increased 10-fold between 1970 and 2000 (1,2). We find portion distortions in restaurants, where the jumbo-sized portions are consistently 250% larger than the regular portion. We even find portion distortions in our homes (3), where the sizes of our bowls and glasses have steadily increased and where the surface area of the average dinner plate has increased 36% since 1960 (4). And if our bowls, glasses, and plates do not distort us, our recipes will. In the 2006 edition of the *Joy of Cooking*, the serving size of some entrées has increased by as much as 42% from some recipes in the first edition of 1931.

This commentary addresses four questions: (a) Who is portion-size prone? (b) Why do portion sizes lead us to overeat? (c) What caused portion distortion? and (d) How can we downsize our consumption norms?

## WHO IS PORTION-SIZE PRONE?

Carefully controlled studies of portion size have been conducted with populations as diverse as economically disadvantaged, poorly educated children to affluent, highly educated professors (5,6). Recent studies even suggest that portion distortion begins as early as 3 years of age (7-9). Whether a child or an adult, whether a dieter or a nondieter, whether an American or a European, whether a 5th grader from Nova Scotia or a world-renowned nutrition researcher, the impact of portion size

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on intake is more similar than different: people tend to eat more from larger-sized restaurant portions (in the general range of 30% to 50% more) (10) and they tend to serve themselves, and eat more from larger-sized packages (in the general range of 20% to 40% more) (11).

When it comes to biasing how much food a person eats, portion size is no respecter of person, position, or profession. Although more affluent, informed, resolved individuals may be more able to avoid buying the larger size to begin with, once it is in front of them, or once they begin to serve themselves onto a 14-in plate, dozens of studies show they behave more similarly than not.

This was vividly illustrated in our recent study in the *American Journal of Preventive Medicine*, in which 85 graduate students and nutrition science professors—many of them internationally renowned—were invited to an ice cream social to share in a celebration for a colleague. On their arrival, they were given either a medium (17 oz) or large (34 oz) ice cream bowl and a medium (2 oz) or large (3 oz) ice cream scoop. Although they were not economically disadvantaged, uninformed, or uneducated, the experts who were given large ice cream dishes served themselves 31% more ice cream. Furthermore, those given the combination of the large dishes and the large scoops served themselves 53% more ice cream than those given small bowls and small scoops (12).

Although studies in the 1970s suggested that obese people might be more portion-size prone than less-obese people, most of the studies since then have shown that portion size influences people of all weights. Some recent studies, including the one published by Colapinto and colleagues in this issue of the *Journal* (13), suggest that income may relate to size-based preferences, but there is no evidence that lower-income people are more prone to overserving themselves from larger packages or overeating from larger portions than higher-income people with similar levels of dietary restraint. Package size, serving size, and dishware size all influence how much all of us eat.

Not only is portion size no respecter of person, it also seems to have no regard for whether a person is hungry or whether they even care for the food. During one study in a Chicago suburb, moviegoers who had just finished lunch were randomly given free medium-size or large-size buckets of stale popcorn to eat during their movie. Although they were not hungry and although the popcorn was stale, they ate 51% more popcorn from the large than the medium buckets (14). This was repeated in a Philadelphia suburb using 14-day-old popcorn at a nighttime movie. Again, although they were not hungry (they had eaten dinner before the movie) and although the food was

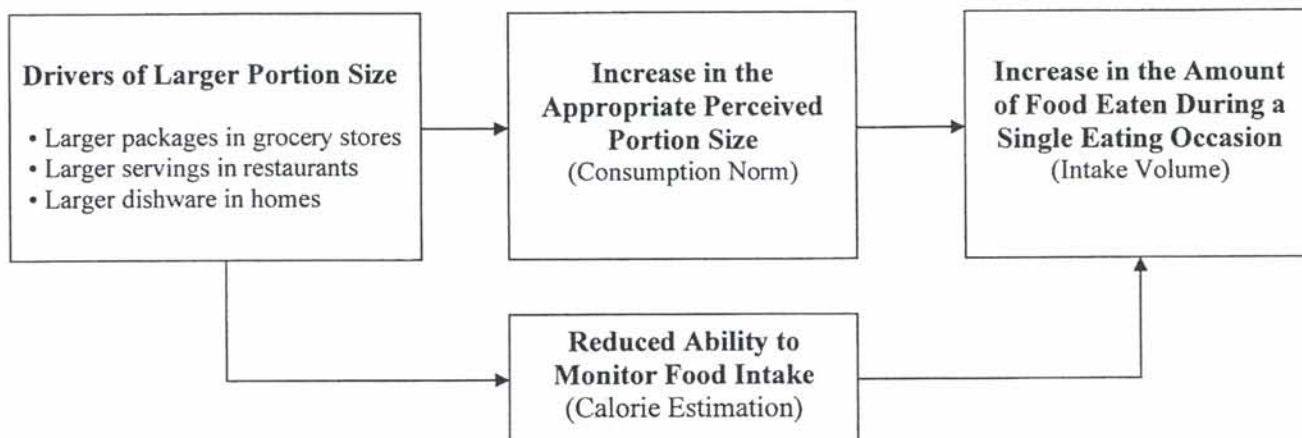


Figure. Why portion sizes lead us to overeat.

not good, those moviegoers given the large buckets ate 35% more of the stale, 14-day-old popcorn (15).

#### WHY DO PORTION SIZES LEAD US TO OVEREAT?

It has often been suggested that we overeat from larger portions because we have a tendency to “clean our plate” (16). Although this may seem to *describe* why many people eat what they are served, it does not *explain* why they do so or why they may have overserved themselves in the first place. The Figure suggests two reasons why portion size may have a ubiquitous, almost automatic influence on how much we eat. First, portion sizes create our consumption norms. Second, we underestimate the calories in large portion sizes.

#### Portion Sizes Create Our Consumption Norms

People can be very impressionable when it comes to how much they will eat. There is a flexible range as to how much food an individual can eat (17), and one can often “make room for more” (18). For this reason, if a person generally eats 8 oz of pasta for dinner, he or she may be quite content eating 6 to 10 oz of pasta for dinner without feeling either overly hungry or overly full.

A key part of the Figure is the role of *consumption norms*. For many individuals, determining how many ounces of pasta to serve themselves for dinner is a relatively low-involvement behavior that is a difficult nuisance to repeatedly and accurately monitor. As a result, people tend to rely on consumption norms to help them determine how much they should consume. Food-related estimation and consumption behavior can be based on how much one normally buys or normally consumes (19). Yet consumption can also be unknowingly influenced by other norms or cues that are present in the environment. An important theme of this commentary is that larger packages in grocery stores, larger portions in restaurants, and larger kitchenware in homes all suggest a consumption norm that very subtly influences how much people believe is appropriate to eat.

Large-sized packages, large-sized restaurant portions, and large-sized dinnerware all have one thing in com-

mon. They all perceptually suggest to us that it is more appropriate, typical, reasonable, and normal to serve and to eat more food than smaller plates or smaller packages would instead suggest (20). These all implicitly influence our personal consumption norm for that situation. This use of consumption norms, as with normative benchmarks in other situations, may be relatively automatic and may often occur outside of conscious awareness (21).

This is what makes these norms so powerful. Even when made aware of them, most people are unwilling to acknowledge that they could be influenced by something as seemingly harmless as the size of a package or plate. Even when shown that larger packages and plates lead them to serve an average of 31% more food than matched control groups, 94% of the diners in four of our field studies resolutely maintained that how much food they served and ate was not influenced by the size of package or plate they had been given (22).

#### We Underestimate the Calories in Large Portions

The second key part of the Figure is the role of *consumption monitoring*. When people pay close attention to what they eat, they tend to eat less. Unfortunately, large portion sizes can either bias people or confuse their estimate of how much they have eaten.

Not surprisingly, how much we end up eating in a distracting environment is partly determined by whether we pay attention to (or attempt to monitor) how much we eat (23). In lieu of monitoring how much we eat, we can instead use cues or rules of thumb (such as eating until a bowl is empty) to gauge how much is the appropriate amount for us to consume. Unfortunately, using such cues and rules of thumb can yield biased estimates and unexpected surprises. In one study, unsuspecting diners were served tomato soup in bowls that were being refilled from tubing that ran under the table, through the table, and into the bottom of the bowls. People eating soup from these “bottomless” bowls ate 73% more soup than those eating from normal bowls, but they estimated that they ate only 4.8 calories more (24).

Our inability to monitor or estimate how many calories we eat becomes increasingly less accurate as portion sizes

increase. It used to be believed that obese people were worse at estimating the calories in their meals than normal-weight people (25). This was even believed to be a contributing cause of their obesity (26). Our recent findings in the *Annals of Internal Medicine* have instead shown that this apparent bias is caused by the size of the meals, not the size of people (27). All people of all sizes—even registered nurses and dietitians—are inaccurate at estimating the calories from large portions (28). Although it initially seems that heavier people are worse estimators of what they eat, they are just as inaccurate at estimating a 2,000-calorie lunch as are their normal-weight colleagues. The answer is meal size, not people size.

With any large-size portion of food—such as a large bag of potato chips—a lot of calories can be eaten before there is any noticeable difference that the supply has decreased. It does not matter how accurate or how diligent a person is at estimating calories, larger portions can obscure any such changes, leading people to eat past the point where they wish they would have stopped.

#### WHAT CAUSED PORTION DISTORTION?

Portion distortion in American restaurants seems to have caught momentum in the late 1970s (2). This coincided with an increasing geographic saturation of fast-food franchises and its accompanying proliferation of inexpensive, convenient dining options (29). With a multitude of different places where one could quickly purchase a hamburger, soft drink, and french fries, an easy way for an ambitious franchise to position itself in the foodscape was by giving more food for only a small increase in price. Given the low food costs of french fries and drinks, they were natural targets for size escalation. Value sizes led to value meals, and value meals at one restaurant led to larger value meals at the restaurant across the street.

History repeated itself 20 years later with the mid-level casual dining restaurants. As their popularity and rivalry increased in the 1990s, so did their attempts to create perceptions of value. They too began trying to differentiate themselves by offering more and more food for less and less money.\*

Do these restaurants super-size their portions because they want us to become fat? No restaurant is specifically in business to make you fat. If you buy a super-sized meal and throw half of it away, it will make no difference to them or to their profitability. They are in business to sell you food, not to make you clean your plate.

The same portion distortion we see in restaurants can be found with the wide range of sizes offered in supermarkets. With the introduction of less expensive generic

*\*Why do we not see the same degree of portion distortion in other countries, such as in Europe? One explanation has to do with the density of restaurant franchises in America versus abroad. Without the high density of “me too” franchises, European restaurants are more highly differentiated from each other. As a result, there is less attempt for consumers to try and compare them using a common denominator such as the size of the hamburger or the number of fries per Euro.*

products, the managers of many popular brands realized that the best way to compete was not through price wars, but through size wars. They could maintain their exclusive positioning, but “add value” by offering larger sizes that cost progressively less per ounce. This resulted in huge boxes of cereal and even double-packs that are often found in warehouse club stores. Our desire for value at the cash register led to larger sizes that cost us less and less.

The same super-sizing that has happened with serving sizes in restaurants and with package sizes in grocery stores also has been happening in our homes (30). As plate sizes, bowl sizes, and glass sizes have increased, by 36% in some cases, so have our assumptions of how much we need to eat and drink to be full (4). Escalations in dishware size lead to a dutiful escalations in how much people see as appropriate amounts to eat. Whereas 6 oz of pasta on an 8-in plate looks like a reasonable portion, the same 6 oz on a 12-in plate would look more like an appetizer. As a result, the typical person would add more pasta to the plate—as the research editorial in this issue of the *Journal* emphasizes (31).

Portion distortion was a predictable economic consequence of people wanting more value for their money (32). What becomes concerning is that it may gradually increase our own internal level of how much we think we need to eat before we are full (33). As 4-oz hamburgers paved the way for 5-oz and 6-oz burgers, so may our adaptation level of how much we think we need to eat in other contexts before we will be full. A real danger of portion distortion is how it may exaggerate how much we think we need to eat when eating at home.

#### HOW CAN WE DOWNSIZE OUR CONSUMPTION NORMS?

Most of us joined the American Dietetic Association because we believe in the importance of nutrition and we believe in the importance of education. The problem with the portion sizes suggested by large packages, large restaurant servings, and large dinnerware is that education is not the answer. Knowing we will serve and eat more will not prevent us—or our clients—from serving more and eating more.

Two years ago I spent 90 minutes explaining to 65 intelligent, motivated graduate students that if I presented them with a 1-gallon serving bowl of Chex Mix (General Mills, Inc, Minneapolis, MN) they would serve themselves and eat more than if I instead presented them with two half-gallon bowls. To make this point vividly clear, I lectured to them, showed them videotapes, asked them to go through a demonstration, and separated them into four-person groups to discuss strategies that they could use to prevent this from happening. In addition to being motivated and intelligent, they were now educated—highly educated—on one topic: If presented with a 1-gallon serving bowl of Chex Mix, they will serve themselves and eat more than if they were instead presented with two half-gallon bowls.

Six weeks after their 90-minute coaching session, I invited these same students to a Superbowl party at a local sports bar, complete with free snacks. On arriving, half were led to one room where they were presented with 1-gallon serving bowls of Chex Mix, and the other half were led to a different room where they were presented

with twice that number of half-gallon serving bowls of Chex Mix. Although intelligent, motivated, and highly educated about the danger of large serving bowls, those people serving themselves from the 1-gallon bowls served themselves 53% more Chex Mix and ate 92% of what they served (34). When asked at the end of the evening if the size of the serving bowl influenced how much they took, all but two denied the possibility.

Is education the answer? The answer is not in telling clients to remind themselves not to overeat from large packages, large servings, and large dinnerware. The answer is for them to eliminate large packages, large servings, and large dinnerware from their lives. It is much easier for a person to change his or her environment than to change his or her thinking.

This can be done painlessly and in small steps. A shopper can buy smaller sizes, or create his or her own single-portion servings by subdividing the bargain-size bag into smaller ones. A restaurant diner can split the fries, order two appetizers instead of an entrée, or have half the dinner packed to go. A home diner can replace large tableware with smaller plates, bowls, and glasses and can use smaller serving bowls and serving spoons, while also keeping the large packages or containers off the table and out of sight.

Just as larger portions have gradually led to super-sized appetites, smaller portions may gradually lead to downsized appetites. We first need to change our personal environment. Only then do we change our minds.

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